# An ERP investigation of food cue responsivity in non-obese, weight gain prone individuals

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## Abstract

An ERP investigation of food cue responsivity in non-obese, weight gain prone individuals
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Obesity is highly prevalent in our society and is challenging to treat in the long term. Weight gain prevention programs are necessary to preclude obesity development, but would be facilitated by a better understanding of who is the most likely to gain weight. A history of dieting with the intent of weight loss has been shown to be a robust predictor of future weight gain, although the mechanisms responsible for this relationship are unclear. One potential factor in propensity towards weight gain is the nature of people's reactions to the abundance of highly palatable food cues in the environment. Event Related Potentials (ERPs) have unearthed differences in how the brain processes palatable food cues among obese and normal weight individuals, as well as restrained and unrestrained eaters. However, comparisons by weight status lack information about whether or not differences are a function of weight gain itself, and restrained eating status has not consistently been found to predict future weight gain. The present study compared ERP response to food cues in non-obese historic dieters (HDs) to non-obese never dieters (NDs). A dieting history by hunger interaction was seen on mean amplitude for P1, N1, P3, and LPP ERP components, suggesting that physiological hunger state has a differential effect on the way HDs and NDs process visual food cues. Specifically, we found that NDs consistently exhibit a larger attentional response when full than when hungry. HDs, on the other hand, show an early, pre-conscious response unaffected by hunger state and a late, sustained response that is larger when hungry than when full.

Future research should test food cue responsivity as a moderator between dieting and future weight gain in order to ultimately better identify those most at risk for weight gain.

# Chapter 1: Introduction

## 1.1. Obesity and obesity proneness

### 1.1.1. Obesity epidemic

The prevalence of obesity in developed countries has been steadily rising. Obesity is defined as having a body mass index (BMI) greater than or equal to 30 $kg/m^2$. A recent report from the Centers for Disease Control and Prevention estimates that 35.7% of U.S. adults and 16.9% of U.S. children are currently obese and another 33.1% of adults and 14.9% of children are overweight (Flegal, 2012; Ogden, 2012). Obesity puts people at a higher risk for heart disease, stroke, type 2 diabetes and certain types of cancer, some of the leading causes of preventable death. Estimates place economic costs of obesity and overweight at $78.5 billion in 1998, more than half of which was covered by Medicare and Medicaid (Finkelstein, 2003), and have been rising since then, with total costs estimated at $147 billion per year in 2008 (Finkelstein, Trogdon, Cohen, & Dietz, 2009).

### 1.1.2. Causes of obesity

On the most straightforward level, obesity is caused by consuming more energy than one expends. This energy imbalance occurs frequently due to a wide variety of biological, behavioral, and environmental factors. Obesity is highly heritable, at levels similar to many other common diseases like cancer and heart disease (Stunkard et al., 1986). While genetic makeup certainly accounts for much of the individual differences in BMI, it cannot solely explain the rapid rate of increase in population BMI over the past decades (Marti, Moreno-Aliaga, Hebebrand, & Martínez, 2004).

Behaviorally, there has been a trend towards decreased physical activity as jobs become more sedentary, automotive transportation gains popularity, and people spend

more time watching TV, on computers, or playing video games. At the same time, eating highly palatable, cheap, and highly caloric foods has become increasingly common. These changes are in large part attributed to our increasingly obesogenic environment (Egger & Swinburn, 1997). The environment can be considered on both a micro and macro level. One's micro level environment consists of foods and opportunities for physical activity available in the home, at school and/or work. The foods kept in one's home and work environment have been shown to be associated with weight status (Larson, Neumark-Sztainer, Hannan, & Story, 2007; Larson & Story, 2010). The availability of supermarkets or convenience stores in the neighborhood is also important in determining the types of food people are likely to purchase. Additionally, opportunities for physical activity in one's neighborhood, including presence of sidewalks, safety, recreational activities, and proximity to one's job, will clearly affect one's decision to exercise. The macro environment includes factors such as government policies surrounding farm subsidies and taxing, as well as food marketing, which less directly influence one's food choices. The obesogenic environment has interacted with genetic predisposition to increase the prevalence of obesity so rapidly.

### 1.1.3. Prevention of weight regain

While some people are successful at losing weight in the short term, maintaining significant weight loss is much more challenging and only a small percentage of individuals who lose weight are able to keep it off (Sarwer, von Sydow Green, Vetter, & Wadden, 2009; Turk, Yang, & Hravnak, 2009). Bariatric surgery is a viable option for the morbidly obese (BMI greater than or equal to 40kg/$m^2$) to lose large amounts of weight and in many cases keep it off for many years (O'Brien, MacDonald, Anderson,

Brennan, & Brown, 2013). However, the majority of people trying to lose weight does not qualify for bariatric surgery or choose it if they do, and success rates in sustained weight loss using behavioral approaches are low. Weight regain has been attributed to several causes. Physiologically, the body responds to weight loss with mechanisms aimed at preventing starvation, including reductions in resting energy expenditure (Johannsen et al., 2012), decreased leptin secretion, and increased ghrelin secretion (Kotidis et al., 2006). Additionally, the environment promoting highly caloric food and low levels of exercise can threaten individuals' ability to continue with weight loss maintenance in the long run.

The scope of health problems associated with obesity, the tendency for a higher weight to persist, and the difficulty of treating obesity successfully makes weight gain prevention critical. However, prevention is also challenging (Stice, Shaw, & Marti, 2006). In a recent review article of studies targeting weight gain prevention in young adults, Laska, Pelletier, Larson, and Story (2012) found that while the transition from adolescence to adulthood is an integral time to target individuals (Nelson, Story, Larson, Neumark-Sztainer, & Lytle, 2008), the evidence for successful preventive efforts is lacking. Similarly in reviews of interventions targeting children and older adults, no consensus on the best strategies has been reached (Brown et al., 2009; Waters et al., 2011). While effective techniques and dissemination methods need to be further developed, another feature of prevention that requires further research is identification of individuals prone to future weight gain.

Even in this pervasive obesogenic environment, not everyone becomes obese. Those individuals who manage to stay in the normal weight range likely have different

responses to the food environment than do those who become obese, or may be biologically protected against weight gain due to a faster metabolic rate or a well-regulated appetite (Bouchard, 2008). However, a particular difficulty in identifying the features that make people prone to obesity is that cross-sectional studies examining differences between obese and normal weight individuals are unable to distinguish between causes and consequences of weight gain. Thus it is imperative to study individuals who are still in the normal weight range to determine what makes some of them likely to gain weight in the future. In order to develop prevention techniques that target individuals prone to weight gain, differential responses to the food environment must be identified pre-morbidly. Identifying predictors of future weight gain could also be very useful in determining the specific biological and behavioral vulnerabilities that create a predisposition toward weight gain.

#### 1.1.4. Predictors of future weight gain

Current weight status is a predictor of future weight status; higher BMI predicts increased likelihood of weight gain later on (Salbe, Weyer, Lindsay, Ravussin, & Tataranni, 2002). Parental weight is also significantly associated with one's chances of becoming obese (Salbe et al., 2002; Stettler et al., 2000; Stice, Presnell, Shaw, & Rohde, 2005; Whitaker, Wright, Pepe, Seidel, & Dietz, 1997). This is in large part due to shared genetics (Stunkard et al., 1986), but environmental factors in childhood may also play a role (Faith, Rha, Neale, & Allison, 1999). Additionally, weight suppression, or the difference between one's current weight and highest weight since reaching adult height, has been found to be a significant predictor of future weight gain (Herzog et al., 2010; Lowe, Davis, Lucks, Annunziato, & Butryn, 2006; Lowe, Annunziato, et al., 2006; Stice,

Durant, Burger, & Schoeller, 2011). A history of being at a higher weight increases the likelihood of gaining back that weight in the future.

Research also suggests that having a very healthy metabolic profile initially (e.g. above average insulin sensitivity for one's weight) may make one more vulnerable to future weight gain (Sigal et al., 1997; Swinburn et al., 1991). As weight increases, these metabolic factors eventually reverse (e.g. one develops insulin resistance), becoming consistent with the poor metabolic profile that is cross-sectionally associated with obesity. The contrast between the metabolic profile of weight gain prone and already obese individuals highlights the importance of identification of premorbid risk factors for weight gain.

A history of dieting to lose weight has also repeatedly been shown to be a significant predictor of weight gain. While this finding could be interpreted as counterintuitive, since presumably aiming to losing weight would result in a lower weight, the research supports the opposite trend. In fact, a recent review of prospective studies (Lowe, Doshi, Katterman, & Feig, 2013) found that those who self reported current or past dieting gained more weight over time than those who did not report dieting in 15 out of the 20 analyses reviewed. Early research of restrained eating assumed that restraint measures were capturing individuals making weight loss efforts as well. However, a closer look has discovered meaningful differences between restrained eating and a history of dieting (Lowe, 1993; Williamson et al., 2007). Measures of restraint, including the Revised Restraint Scale (RRS), the restraint subscale of the Three Factor Eating Questionnaire (TFEQ-RS), and the Dutch Restrained Eating Scale (DRES) (Herman & Polivy, 1980; Stunkard & Messick, 1985; van Strien, Frijters, Bergers, &

Defares, 1986) do not directly assess motivations aimed at weight loss. Rather, they capture a desire to avoid or reverse weight gain (Chernyak & Lowe, 2010). The RRS identifies individuals who tend to have a long history of dieting and weight fluctuations; nonetheless most restrained eaters are not currently dieting (Lowe, 1993). Laboratory studies have supported the distinction between restrained eating and current dieting. Individuals who are currently on a diet respond differently to eating a high calorie preload than do those who are not currently dieting. Current dieters eat less following a preload than they do without a preload, while restrained non-dieters tend to eat more following a preload than without a preload (Lowe, Whitlow, & Bellwoar, 1991; Lowe, 1995). Giesen, Havermans, Nederkoorn, Strafaci, and Jansen (2009) compared unrestrained non-dieters, restrained non-dieters, and restrained current dieters on a task where they were asked to play a game to earn points either towards snack food or fruits and vegetables. The RRS was used to determine restraint level. Participants were told they would consume the amount that they "earned" following the game. Restrained eaters worked harder to earn snack food than unrestrained eaters did, but only if they were not currently dieting; current dieters put forth the least amount of effort to earn snack food.

Guerrieri, Nederkoorn, Schrooten, Martijn, and Jansen (2009) also compared these three groups and similarly found important effects of current dieting status. They experimentally induced either impulsivity or inhibition, and then asked participants to participate in a sham taste test. While induced impulsivity increased consumption in unrestrained and restrained non-dieters compared to induced inhibition, the opposite pattern was seen for current dieters. They in fact decreased consumption following the impulsivity condition compared to the inhibition condition. As further evidence for the

difference between dieting and restraint, restrained eating, unlike current or past weight loss dieting, has not been found to predict future weight gain in the majority of cases (Lowe et al., 2013). Therefore, the goal of losing weight (i.e., current or past dieting) rather than avoiding weight gain (i.e., being a "restrained eater"), appears to be a critical component of dieting that is associated with future weight gain. Restrained eating is best characterized as a conscious effort to consume less food than desired, which may or may not co-occur with weight loss dieting.

The evidence is not clear on whether chronic dieters are biologically and/or behaviorally prone to weight gain to begin with, if the act of dieting itself causes future weight gain, or if dieting behavior is merely correlated with some other variable that contributes to weight gain and obesity. One longitudinal study of adolescents found that the relationship between dieting history and weight gain was partially mediated by increased binge eating and decreased breakfast consumption (Neumark-Sztainer, Wall, Haines, Story, & Eisenberg, 2007), indicating that dieting may be associated with unhealthy eating behavior that could contribute to the weight gain. Repeated dieting has also been found to biologically alter the way in which food is processed in the future, potentially contributing to weight gain (Dulloo, Jacquet, & Montani, 2012). However, the evidence that dieting history is simply a proxy for identifying those otherwise prone to weight gain is also strong (Lowe & Levine, 2005). In the study mentioned above, individuals who tend to engage in binge eating and decreased breakfast consumption might have done so regardless of dieting history. A predisposition toward weight coming from the other sources, such as genetic predisposition, weight suppression, and increased appetitive drive, leads to overconsumption and weight gain. Dieting is a natural response

to weight gain, but is usually not effective at reducing weight in the long term. A history of dieting might be a simpler way to identify these individuals with an increased susceptibility for weight gain.

Regardless of the mechanism, dieting history appears to be an excellent indicator of weight gain proneness and can be used to premorbidly identify those at risk. In order to better understand this predictive relationship, it would be useful to examine how non-obese historical dieters, that is, individuals with a history of dieting with the intent of weight loss and with a BMI below 30 $kg/m^2$, respond to environmental food stimuli in comparison to non-obese never dieters, those who have never gone on a diet to lose weight. Restricting the comparison to non-obese individuals is important in order to target the role of dieting in obesity prone, rather than already obese, individuals. All non-obese individuals with a dieting history, regardless of current dieting status, are hypothesized to be at increased risk for weight gain. However, the expression of this latent predisposition towards weight gain may depend on current dieting status. If a historical dieter is not currently on a diet, the underlying predisposition will tend to be expressed in physiology and behavior. If they are currently dieting, their behavior may be incongruous with their natural tendencies, and the expression of their predisposition may in fact be reversed in the short term (Giesen et al., 2009; Guerrieri et al., 2009; Lowe et al., 1991; Lowe, 1995). Current dieters may be able to inhibit their desire to eat palatable foods that violate their dietary rules in the short term, but eventually, once their diet ends, their natural tendency towards excessive consumption and weight gain will return. In order to examine mechanistic factors in why frequent dieters gain weight, it is necessary to exclude current dieters. Past dieters are likely to be exhibiting behavior most aligned

with their natural tendencies that result in ultimate weight gain. Including current dieters would contaminate the variable of historical dieting by adding people who are purposefully acting against their trait behaviors.

## 1.2. Measuring response to food cues

### 1.2.1. Behavioral measures of response to food cues

Several behavioral tools have been developed to measure one's level of attention towards a certain cue. The dot probe task (Mogg, Bradley, & Williams, 1995) is commonly used. This procedure involves two images being flashed simultaneously on a screen, one on the left and one on the right. Next a dot flashes on one side, and the participant is instructed to respond with the side where the dot is presented as quickly as possible. When a food image and nonfood image are paired, reaction time when the dot is on the side with the food as compared to the other side can be measured. If reaction time when the dot and food image are congruent is shorter than when they are incongruent, the participant is said to have an attentional bias towards the food cue. Eye tracking is often used in conjunction with the visual probe task, as a noninvasive measure of visual attention to salient cues (Castellanos et al., 2009). Another commonly used measure is the emotional Stroop test. The Stroop asks participants to name the color ink in which a word is written, rather than read the word itself. Response time is expected to increase with an emotionally charged, in this case food-related, word, since it becomes difficult to disassociate from the written word in order to concentrate on the ink color (Calitri, Pothos, Tapper, Brunstrom, & Rogers, 2010). However, discerning which processes these tests are reflecting is difficult, and low correlations between the food-related Stroop and

dot probe indicate that they are likely measuring distinct underlying constructs (Pothos, Calitri, Tapper, Brunstrom, & Rogers, 2009).

### 1.2.2. Neural measures of response to food cues

#### *1.2.2.1 fMRI*

Functional Magnetic Resonance Imaging (fMRI) has been used to identify neural processes involved with food cue biases seen in behavioral studies. Differences in response activity in individuals considered prone to weight gain or obesity, relative to those without this predisposition, have been found in areas such as the hypothalamus, amygdala, hippocampus, prefrontal and orbitofrontal cortex, and insula, brain regions associated with emotional and reward processes (Cornier et al., 2013; Ely, Childress, Jagannathan, & Lowe, 2013; Stice, Yokum, Burger, Epstein, & Small, 2011). fMRI has the benefit of measuring detailed spatial resolution about exactly where activity is occurring in the brain. However, it lacks temporal detail; fMRI measures the change in blood flow associated with brain activation, which takes between 7 and 12 seconds to occur (Heeger & Ress, 2002).

#### *1.2.2.2. ERP*

Conversely, electroencephalography (EEG), the recording of electrical activity along the scalp, has a high level of temporal detail (Woodman, 2010). EEG works by measuring voltage fluctuations resulting from ionic current flows within the brain's neurons (Luck, 2005). As recordings are taken at the scalp level, there is an issue of source localization, meaning they lack the accurate spatial resolution of fMRI. Techniques exist to estimate the location of brain activity based on electrode location, but they are based on rough assumptions. However, the high resolution of temporal

information gathered with EEG is integral in understanding stages of perceptual and cognitive processing, as they occur on a millisecond time scale. Because EEG reflects multiple, simultaneously ongoing brain processes, examining waves over one trial generally does not provide interpretable information about how the brain is processing some specific stimulus. Event Related Potentials (ERPs) use simple averaging techniques of EEG waves over multiple trials in order to understand how perception, processing, and understanding of a stimulus occur. By time-locking EEG waves to a stimulus presentation and averaging them over many trials, extraneous brain activity is averaged out, and the brain's response to the stimulus remains as an averaged waveform, called an ERP (Luck, 2005).

ERP waveforms consist of a series of positive and negative voltage deflections, conventionally plotted with negative going waveforms upward and positive going waveforms downwards. A waveform amplitude is the difference between the voltage of a scalp electrode and a reference electrode. These waveforms reflect multiple underlying latent components, defined as neural activity that arises from some cognitive/affective function. Components can vary in their polarity, that is, whether they are negative-going (N) or positive-going (P); timing following a stimulus; and scalp distribution, because the same cognitive function can occur in multiple brain regions at different times and in different ways (Luck, 2005). They are usually named based on polarity, and either the time in milliseconds following stimulus presentation, or their poststimulus order (Kounios, 2007). Because of the variety and overlapping nature of components, they are very difficult to isolate when analyzing a particular waveform. However, all ERP research involves making estimates of components in order to make inferences about the

cognitive functioning underlying ERPs. Conscious awareness of a visual stimulus has been estimated to occur 200-300 ms following stimulus presentation on average (Wilenius & Revonsuo, 2007).

#### *1.2.2.3. ERP in food cue research*

Most studies examining ERP response to food cues have focused on P3 and LPP components (Gable & Harmon-Jones, 2010; Hill, Wu, Crowley, & Fearon, 2013; Nijs, Franken, & Muris, 2008, 2010; Nijs, Muris, Euser, & Franken, 2010; Nikendei et al., 2012; Stockburger, Schmälzle, Flaisch, Bublatzky, & Schupp, 2009; Svaldi, Tuschen-Caffier, Peyk, & Blechert, 2010; Watson & Garvey, 2013). The P3's name reflects that it most commonly appears as a positive-going wave occurring between 300ms and 500ms following stimulus presentation. It is generally thought of as two separate components: The P3a, seen in frontal electrodes, and the P3b, seen in parietal electrodes. The P3b is the most widely studied ERP component, and has been understood to reflect recognition and updating of memory (Patel & Azzam, 2005). The LPP (Late Positive Potential) component occurs between 500 ms and 800 ms following stimulus presentation and has been shown to reflect explicit task relevance or emotional significance (Cuthbert, Schupp, Bradley, Birbaumer, & Lang, 2000). Emotion and addiction research have provided strong support for an association of the P3 and LPP with emotional content or motivational significance of the visual stimulus presented (Littel, Euser, Munafò, & Franken, 2012; Olofsson, Nordin, Sequeira, & Polich, 2008). They are commonly elicited using "oddball" tasks, in which participants are asked to keep track of certain infrequently presented target stimuli, which occur mixed in with more frequent standard stimuli. In these cases, the task makes certain stimuli significant. The same pattern can be

seen when an emotionally relevant, inherently significant stimulus (such as a palatable food) is presented (Hajcak, MacNamara, & Olvet, 2010). P3 and LPP components are considered to reflect conscious levels of processing, occurring long enough after stimulus presentation that the individual is consciously aware of what they are seeing. There is also evidence that the amplitude of late positive potentials is at least partly under intentional control (Hajcak, Dunning, & Foti, 2009)

A few studies have also studied the role of food cues on the N2 component (Nijs, Franken, et al., 2010; Watson & Garvey, 2013). The N2 refers to a component most commonly negative in voltage, although at times positive (Nijs, Franken, et al., 2010), occurring approximately 200 ms following stimulus presentation, and often maximal over central electrodes. The N2 is most commonly considered to result from a deviation in visual form or context of a stimulus, also usually elicited through an experimental oddball paradigm (Folstein, Van Petten, & Rose, 2008; Patel & Azzam, 2005). However, it may also reflect level of cognitive control or conflict monitoring (Huster, Enriquez-Geppert, Lavallee, Falkenstein, & Herrmann, 2013; Watson & Garvey, 2013), increasing its relevance to food-related studies in which a participant may both be drawn to and motivated to avoid the same food stimulus.

Another potential component of interest is referred to as Early Posterior Negativity (EPN), defined as a negative-going waveform in the posterior region along with a positive amplitude in temporo-occipital sites, most pronounced between 200 and 300 ms (Olofsson et al., 2008; Stockburger, Weike, Hamm, & Schupp, 2008). This component has primarily been found to have significance following emotionally salient stimuli (Schupp, Flaisch, Stockburger, & Junghöfer, 2006), but it has been implicated in

food cue studies as well (Blechert, Feige, Joos, Zeeck, & Tuschen-Caffier, 2011; Stockburger et al., 2009, 2008). It has been theorized to reflect "natural selective attention," identifying features of a visual stimulus that require further processing (Dolcos & Cabeza, 2002; Olofsson et al., 2008). The N2 and EPN both occur around the same time period as conscious awareness begins, and have been theorized to represent preconscious processes that are involved with orienting attention towards salient stimuli. The degree to which they correlate is unknown, but they may both represent parts of an underlying process of selecting task-relevant objects to be processed more fully (Olofsson et al., 2008; Schupp et al., 2007).

### 1.3. Food deprivation and satiety

Because weight gain occurs from eating beyond physiological need, it is critical to take level of physiological satiety into account when considering response to food cues. Level of food deprivation has repeatedly been shown to have a significant effect on behavior surrounding consumption and anticipation of food (Drobes et al., 2001; Plihal, Haenschel, Hachl, Born, & Pietrowsky, 2001; Stockburger et al., 2009, 2008). When in a food-deprived state, the relative value of food increases (Raynor & Epstein, 2003), as does selective attention paid to food cues (Loeber, Grosshans, Herpertz, Kiefer, & Herpertz, 2013; Piech, Pastorino, & Zald, 2010; Tapper, Pothos, & Lawrence, 2010) and approach tendencies towards food cues (Seibt, Ha, & Deutsch, 2007). While increased motivation towards food cues is normative in a hungry state, maintained motivation when satiated may be a sign that environmental or cognitive factors, unrelated to homeostatic hunger, are influencing appetite (Lowe & Butryn, 2007). As weight gain prone individuals by definition must consume more energy than their body requires if they do in

fact gain weight, they may be particularly likely to experience this increased motive to continue eating even when not physiologically hungry (Coletta et al., 2009; Ely et al., 2013).

### 1.4. Similarities to drug and alcohol addiction studies

Theories of how palatable food cues elicit such a salient response parallel those of addictive drug and alcohol behaviors, in that both are based on a model of classical conditioning. Stimuli repeatedly paired with an addictive behavior have been found to trigger drug cravings. For example, in one study cocaine addicts showed increased activation of dopaminergic pathways in the mesencephalon, associated with motivationally salient stimuli, when exposed to words related to drug use as compared to neutral words. This increased activation was not seen in non-users (Goldstein et al., 2009). Additionally, another study found alcoholics to have a larger P3 amplitude in response to alcohol-related images than neutral images, reflecting an alcohol-specific cue-reactivity, a difference not seen in healthy controls (Herrmann et al., 2000). In both of these cases, drug/alcohol addicts experienced increased activation in response to substance-related stimuli, reflecting high motivation towards those items. This is likely due to or exacerbated by the classical conditioning phenomenon previously described.

There is some evidence to suggest that individual differences in reward-related response to salient cues may actually precede future addictive behaviors. Specifically, increased reward region sensitivity in adolescents in response to anticipated monetary reward has been found to predict future substance abuse onset (Stice, Yokum, & Burger, 2013) indicating that certain individuals may have a proneness to become conditioned to substance-related, and perhaps other strongly rewarding, cues early on.

Parallels in neural functioning between drug addiction and obesity have been found, specifically relating to dopamine release in mesolimbic regions, which correlates with reported reward (Volkow, Wang, Fowler, & Telang, 2008). Both drug addicts and obese individuals tend to have hyperresponsive reward reactions to drug or food cues (Kalivas & Volkow, 2005; Stice, Spoor, Ng, & Zald, 2009). Although questions remain about whether "food addiction" exists in the way that drug addiction does, these similarities can inform our thinking about the reasons why some people may be more responsive to food cues than others. Specifically, stimuli related to eating palatable foods become conditioned to trigger a desire to eat, and some people may be at an increased proneness to become conditioned to palatable food cues. Furthermore, individual differences in propensity towards being conditioned might also depend on the state of hunger when conditioning occurs. Strength of conditioning when someone consumes food in a state of physiological hunger may be different than strength of conditioning when someone consumes highly palatable food in a state of physiological repletion.

## 1.5. Food cue responsivity

### 1.5.1 Obese vs. normal weight

Obese individuals, relative to those at a normal weight, may experience a difference in responsivity to palatable food cues, such as pictures, smells, or presence of the actual food, which are pervasive in our obesogenic environment. However, findings in the way in which responsivity differs have been inconsistent. Obese individuals show an elevated drive to seek out palatable food in self-report and behavioral data, and report greater enjoyment of the taste of food that they consume. However, neuroimaging data reveal a more complicated story, with both hyper-responsivity and hypo-responsivity of

reward circuitry being seen depending on study design and brain region examined (Beaver, 2006). Stice, Spoor, Ng, and Zald (2009) present an explanation that accounts for both increased and decreased activity of rewarding brain regions surrounding eating. Based on a comprehensive review, they concluded that obese relative to lean individuals fairly consistently show increased brain activation in both reward and gustatory regions in response to anticipation of intake. In response to consumption, they continue to exhibit increased activation of gustatory regions, supporting the idea that they may receive more pleasure from the food they eat. However, compared to lean individuals they show decreased activation in the dorsal striatum, an area with a high density of dopamine receptors. Obese individuals may have increased reward activation to anticipation of palatable foods, as well as increased enjoyment of the food they consume, but a decreased reward response to actual consumption. This creates a situation where obese individuals are highly motivated to seek out food, but are not as rewarded as normal weight individuals when they consume it, resulting in ongoing motivation to continue eating once they begin. Based on the conclusions of Stice et al (2009), differences in ERP response to food in obese and lean individuals are expected to depend on whether participants are anticipating food intake (e.g. viewing images of palatable foods) or actually consuming food.

ERP has repeatedly uncovered differences in processing of food images in obese compared to normal weight individuals in P3 and LPP components thought to reflect conscious, sustained attention. Babiloni et al. (2009) used an oddball paradigm to compare P3 response in obese and normal weight individuals. The infrequent stimulus was an image identical to the frequent stimulus, but stretched horizontally to be 25%

wider. Face, food, and control (e.g. flower) stimuli were presented. On average, obese participants had a reduced medial prefrontal P3 response to stretched food and face stimuli compared to lean participants, a difference not found with control stimuli. While all participants recognized the novelty of stretched control stimuli, the reduced P3 in obese individuals suggests that only lean participants noticed when face and food stimuli were stretched. Authors hypothesize that the smaller component may reflect a reduced ability to recognize larger portion sizes in obese individuals. Nijs, Muris, et al. (2010) compared normal weight to overweight/obese (BMI: M=30, SD=4.62) females in their P3 response to high calorie snack food versus control items, this time taking hunger level into account. They asked participants to count the number of stimuli shown in a certain category (either food, office items, or babies). All participants, regardless of weight or hunger status, had an increased posterior P3 amplitude to food as compared to nonfood stimuli. However, normal weight participants had a larger posterior P3 in response to food items when hungry compared to when satiated, a difference not seen in the overweight/obese group. Normal weight participants had a larger P3 in response to food cues than overweight/obese participants when hungry, and overweight/obese participants trended towards a larger P3 than normal weight participants when satiated. However, overweight/obese participants ate more in a sham taste test when hungry than normal weight participants did. Because the P3 has been found to at least partially be under the influence of intentional control (Hajcak et al., 2009), authors hypothesize that the decreased amplitude of the P3 in the overweight/obese group when hungry may represent the use of cognitive strategies to avoid sustained attention to palatable food cues (Nijs, Muris, et al., 2010). Another potential explanation for the lack of correlation between

hunger level and attraction towards food cues in obese participants may be an overreliance on external cues, rather than internal signals, in determining consumption (Rodin & Slochower, 1976). Together these two studies indicate that obese individuals tend to respond differently from normal weight individuals to palatable food cues. Their motivation to eat may be based more on the environment than on internal hunger signals, and they also may actively attempt to down-regulate their attention towards a palatable food cue in an attempt to eat less, particularly when they are hungry.

In some cases, no differences in P3 response were seen comparing obese to lean individuals. Nijs et al. (2008) measured P3 and LPP response as obese and normal weight participants passively viewed a series of highly palatable food and non-food stimuli. Hunger was assessed, but not experimentally manipulated. Central and posterior P3 and LPP amplitudes were significantly larger for food than non-food cues, but no between-group differences were found. The participants were next asked to complete a Stroop task (Nijs, Franken, et al., 2010). A mix of food- and office-related words was presented, each time in a different color ink. Participants were instructed to respond to the color ink and ignore what was written. Again, a main effect of word type was found, such that P3 amplitude was larger for food-related words than office-related words in central and posterior regions. However, no group differences were seen. The normal weight group reported higher levels of hunger at pretest than the obese group, which could potentially mask any group differences that may have existed. Additionally, because obese individuals may respond differently to a hunger manipulation than would normal weight individuals, perhaps even in an opposite direction as was seen in results of Nijs, Franken,

et al., (2010), the failure to manipulate hunger may be the reason for the observed lack of between group differences.

This same study (Nijs, Franken, et al., 2010) discovered differences in P2 amplitude between obese and normal weight groups on the emotional Stroop task. Specifically, anterior and central P2 amplitudes in response to food words were larger in obese than normal weight individuals, and only in obese individuals, a marginally significant difference in anterior P2 amplitude was found between food-related and office-related words. While this study found differences in a positive-going wave, rather than the typical negative presentation of the N2 and EPN components, they were seen in the same brain regions during the same time period, suggesting that they may be reflecting similar underlying processes as the N2 and EPN are thought to indicate. Results imply that obese participants allocated more attention towards food words than normal weight participants did in the early, preconscious stages of information processing, but not later on. The preconscious, but not subsequent, effect could again potentially be explained by a volitional attempt to reduce attention towards the food cue once cognitive processes entered the conscious time frame (Nijs, Franken, et al., 2010). These findings highlight the importance of examining preconscious components in order to isolate attentional response from attempts to override their natural response patterns.

### 1.5.2. Food cue responsivity in non-obese individuals

While studies of differences between obese and normal weight individuals provide useful information, they do not reveal the temporal relationship of weight gain and neurocognitive characteristics. In order to understand whether food cue responsivity plays a role in the development of weight gain leading to obesity, individuals must be

studied before the obese state develops. Therefore, comparing responsivity to food cues among groups who are otherwise identified as weight gain prone or resistant, but are not yet obese, would shed light on whether differences in food cue responsivity could help to explain the reasons behind differences in proneness. Although dieting history has emerged as a much more consistent predictor of future weight gain than has restrained eating, the lack of studies focusing on historic dieters indicates a utility in reviewing work comparing restrained and unrestrained eaters as well, as there may be some relevant similarities between restrained eaters (especially those assessed with the RRS) and historic dieters. Also due to limited research in this area, both fMRI and ERP studies will be reviewed. However, the overlap between these two techniques is not fully understood, and their results cannot be directly compared.

#### *1.5.2.1. Restrained eating*

Research on differences in food cue responsivity between restrained and unrestrained eaters has been examined using a variety of paradigms. Restrained eating, or restricting intake in order to prevent weight gain (Lowe & Levine, 2005), has not been found to predict future weight gain in the majority of cases (Lowe et al., 2013), possibly because restrained individuals are employing successful strategies to resist a vulnerability towards overeating. Comparatively, normal weight unrestrained eaters most likely do not have that vulnerability to begin with. One fMRI study found stronger responses in reward-related brain regions to milkshake consumption, but not to anticipated milkshake receipt, in restrained eaters compared to unrestrained eaters as defined by DRES scores (Burger & Stice, 2011), explained as representing a hyper-responsive reward circuitry in restrained eaters. As previously discussed, obese individuals tend to have a stronger

reward response to the anticipation of food than to actual consumption (Stice et al., 2009). Just as having a very healthy metabolic profile, opposite from what is commonly seen in obese individuals, has been shown to predict future weight gain (Swinburn et al., 1991), the reward response pattern of restrained eaters, opposite from the pattern of those already obese, may represent a vulnerability to weight gain. The especially high reward response that restrained eaters experience in response to palatable food consumption would increase the incentive to seek out these foods, although they may be employing strategies to successfully resist this vulnerability.

Watson & Garvey (2013) used ERP to compare restrained and unrestrained eaters, defined with the DRES, on their response to food cues in a Go/No-go task. Participants were instructed to press a button every time they saw a frequently presented green triangle ("go" stimuli) and to refrain from pressing the button when they saw any other image ("no-go" stimuli). The no-go stimuli were divided into high calorie food and non-food images, and were presented less frequently than the green triangle. Go/no-go tasks are frequently used to measure response inhibition and cognitive control, so authors had hypothesized that restrained eaters would have larger P3 and N2 responses to food-related no-go trials, as they would be more emotionally salient, increasing cognitive control required to inhibit a response. Ultimately they found no differences in N2, P3, or LPP responses to food vs. non-food no-go trials between restrained and unrestrained eaters. One limitation to this design is the lack of any reported hunger measure, to either control hunger level or even keep track of it. Manipulating hunger might be the best way to detect differential ERP responses to food.

Another study used ERPs to measure difference in response to food (chocolate)

and non-food (floral) odors in restrained and unrestrained eaters, as defined with the TFEQ-RS (Kemmotsu & Murphy, 2006). In one condition, participants were forced to attend to the odor by rating its magnitude. In the other, they were encouraged to ignore the odor by being asked to engage in a computer task. Findings indicated that unrestrained eaters displayed a larger P3 response to the food odor in the attend condition than the ignore condition, a difference not seen in restrained eaters. Additionally, restrained eaters had a smaller N2 component in response to the food odor than unrestrained eaters did. These differences could be explained by a cognitive suppression of food-related attention and response in restrained eaters. Their ability to suppress their response may override the conscious attention towards the odor required to complete the study task. As restrained eaters defined by the TFEQ-RS are not generally prone to future weight gain, this early suppression of attention may be a successful strategy to prevent overeating.

These studies vary significantly in their methodology and procedure. However, none found differences between restrained and unrestrained eaters in their response to visual food cues without actual food receipt. Differences were seen when a food odor, rather than image, was presented, suggesting that odor may be a particularly salient cue. Because restrained eaters as defined by either the TFEQ-RS or DEBQ are not at increased risk for future weight gain, the lack of significant differences in response to food cues between restrained and unrestrained eaters is not surprising. The type of restrained eater as defined by the RRS is more similarly aligned to weight-gain prone chronic dieters who may demonstrate a higher responsivity to environmental food cues.

#### *1.5.1.2. Weight gain proneness*

Weight gain proneness can be measured in multiple ways, as previously discussed. Because the research is so limited in comparing response to food cues in weight gain prone and resistant individuals, restrained eaters as defined by the RRS will be included in this category as well, since a high RRS score reflects frequent dieting in the past. However, it is important to note that authors of these studies were not conceptualizing groups based on weight gain proneness. Rather, they viewed the chronic dieting of restrained eaters itself as the cause of the appetitive problems this group demonstrates. Five fMRI studies have compared passive viewing of food images in weight gain prone to weight gain resistant individuals (Coletta et al., 2009; Cornier et al., 2013; Demos, Kelley, & Heatherton, 2011; Ely et al., 2013; Stice, Yokum, et al., 2011). In one case (Cornier et al., 2013), obesity proneness was defined by self-reported struggles with weight, at least one obese first degree relative, and a BMI between 20 and 30 $kg/m^2$, as compared to self-reported naturally lean individuals who had never been overweight, with a BMI between 17 and 25 $kg/m^2$ and no obese first degree relatives. While hedonic food images as compared to nonfood images created an increased response in reward-related brain regions (insula, inferior prefrontal cortex, and medial prefrontal cortex) in all participants when hungry, the increased response to hedonic food cues remained only in obesity prone individuals when full. In fact, obesity prone individuals had increased activity in the insula and inferior prefrontal cortex when viewing palatable foods following a meal. If the reward response does not decrease after a meal, the obesity prone individual presumably would still find food cues appealing and would therefore be more vulnerable to continue eating and eventually gain weight. They

may even be more driven to continue eating palatable foods after they have already eaten, compared to when they are hungry (Cornier et al., 2013; Lowe & Levine, 2005)

Another study defined weight gain prone and resistant adolescents based only on parental weight status, not the adolescent's own struggles with weight. They found greater activation in reward-related brain regions in obesity prone compared to resistant adolescents during actual food consumption, but no differences during anticipation of consumption (Stice, Yokum, et al., 2011). Authors explained this finding as a gradual shifting of reward sensitivity from receipt of food reward towards conditioned stimuli associated with repeated pairings with food receipt for obese-prone adolescents (Stice et al., 2009). Those at highest risk for weight gain demonstrate the reverse pattern of reward activation than do those who are already obese, consistent with findings regarding metabolic processes (Swinburn et al., 1991).

The other three studies defined weight gain proneness by only considering a history of dieting. Ely et al. (2013) defined historic dieters as having at least two intentional past weight loss efforts and not currently being on a diet, and non-dieters as those with no history of weight loss dieting. Activation of reward circuitry when viewing palatable food cues was measured in fasted and fed states. While no differences between groups were seen in the fasted state, historic dieters had bilateral increased activation of the prefrontal cortex following a meal compared to non-dieters, again suggesting that eating does not attenuate the rewarding effect of palatable food cues in obesity-prone individuals, leaving them more susceptible to overeating and weight gain. Demos et al. (2011) found a similar pattern; chronic dieters, as defined by the RRS (which reflects dieting history), showed higher reward-related activity to palatable food cues after receipt

of a high calorie milkshake than after receipt of water, whereas the opposite pattern was seen in non-dieters, categorized by low RRS scores. Finally, Coletta et al. (2009) compared response to palatable food cues in those with high and low RRS in both a fed and fasted state. Significant differences were seen between groups, such that in the fasted state, non-dieters had higher activation than chronic dieters in brain regions related to hunger, expectation of reward, and reinforcement. However, in the fed state, chronic dieters, but not non-dieters, had increased activation of reward and hunger-related regions. Together, these studies provide strong evidence for the idea that, following a meal, weight gain prone individuals do not experience the attenuation of rewarding value of food cues that weight gain resistant individuals do; indeed they experience an increase in reward response. The draw of food cues may in fact rise after eating, increasing likelihood of eating beyond physiological need.

ERP has only been used to study differences between weight gain prone and resistant individuals in one case so far. Blechert, Feige, Hajcak, and Tuschen-Caffier (2010) used the RRS to categorize participants as restrained or unrestrained. The "restrained" group with a history of chronic dieting weighed significantly more than the non-dieting group (BMIs of 24 and 20, respectively). They had them perform two experiments; in the first, participants passively viewed images of palatable food, pleasant non-food items, unpleasant non-food items, and neutral items. No between-group differences in ERP response were apparent. In the second experiment, participants were shown food items from two menus. They were told that they would be able to consume the food listed on one menu following the experiment, but not the other menu. Chronic dieters and non-dieters responded differently to this availability manipulation in that

chronic dieters' central posterior LPP amplitudes in response to the available foods were smaller relative to the unavailable foods, a difference not seen in non-dieters. Authors hypothesized that the smaller amplitude could be due to a down-regulation of attention towards available food, in an active attempt to prevent overeating when the food would be later presented. Because weight was not included as a covariate in analyses, differences could potentially be due to BMI rather than dieting history. Although participants were asked to eat a meal three hours before their study visit, hunger was not experimentally controlled, and reported hunger levels varied. Additionally, current dieting status was not taken into account. While the use of ERP to compare weight gain prone and resistant individuals in their response to food cues thus far has been limited, the superior temporal resolution as compared with fMRI could provide a finer-grained analysis of where, in the process of perceiving and attending to food stimuli, brain response differs in weight gain prone and resistant individuals. In particular, the time course of food cue processing could provide insight into whether weight gain prone individuals show more reactivity to these cues in the early, preconscious stages of processing. Perhaps they are reacting more strongly before they even consciously recognize what they are seeing.

### 1.6. The Present Study

The extant literature leaves much work to be done on the role of historic dieting on response to food cues. Because of the wide variety in methodology and sampling in the reviewed studies, no single conclusion can be drawn about differences in brain response to food cues in individuals prone to weight gain. One major limitation to the current literature is the frequent use of restrained eating scales to define individuals who

exert more effort to control their food intake. Historic dieting has been found to predict future weight gain much more consistently than restrained eating but measures of restrained eating have been used much more frequently to document appetitive abnormalities such as counterregulatory eating.

Two discrepant explanations of findings tend to be hypothesized by authors of these studies. Studies that found an increased response (be it fMRI response in reward-related brain areas, a larger pre-conscious ERP component, or a larger late ERP component) to food cues in people with a tendency towards weight gain (in some cases already obese, in some cases restrained eaters, and in some cases chronic dieters), explain this finding as a sensitivity or increased attention towards palatable food, leading to a higher likelihood of overconsumption (Babiloni et al., 2009; Coletta et al., 2009; Cornier et al., 2013; Demos et al., 2011; Ely et al., 2013; Nijs, Franken, et al., 2010). On the other hand, some studies found a decreased response to food cues in people with a tendency towards weight gain. These have been explained in some cases as a purposeful down-regulation of attention to salient food cues in order to avoid overeating. In others they have been thought of as a demonstration of opposite responses between already obese and weight gain prone individuals (Blechert et al., 2010; Burger & Stice, 2011; Kemmotsu & Murphy, 2006; Nijs, Muris, et al., 2010; Stice, Yokum, et al., 2011; Swinburn et al., 1991).

The present study aimed to study a new model of weight gain proneness based on historic dieting to determine if this conceptualization leads to more consistent results. Only females were included based on prior research (Demos et al., 2011; Ely et al., 2013), because females and males tend to have different patterns in how and why they

gain and lose weight (Holm-Denoma, Joiner, Vohs, & Heatherton, 2008), and because it was not possible to include enough male and female participants to meaningfully examine potential sex effects. Age was limited to young adults (18-30) because of our easily accessible undergraduate student population. Because of the consistent findings that a history of dieting predicts future weight gain, dieting history was the principal variable for defining individuals as either prone or resistant to weight gain. Specifically, weight gain prone individuals must have gone on at least one weight loss diet in the past two years, and must not have been currently dieting to lose weight. This group was called historic dieters (HDs). Non-weight gain prone individuals must have never gone on a diet to lose weight. They were called non-dieters (NDs). An important aspect of HDs in the present definition is the lack of a current diet. Individuals currently dieting to lose weight were excluded from participation due to research demonstrating that restrained eaters currently on a diet to lose weight tend to behave differently than do restrained eaters not currently dieting to lose weight (Giesen et al., 2009; Guerrieri et al., 2009; Lowe et al., 1991; Lowe, 1995). In order to isolate the natural behavior of chronic dieters as accurately as possible, those currently dieting were not included.

Because of the importance of identifying predictors of weight gain premorbidly, participants were limited to those in the normal weight and overweight range (i.e. BMI between 20 $kg/m^2$ and 30 $kg/m^2$) so as to compare those prone to weight gain to those who are not prone, without potential confounds that arise from studying those who are already obese. While BMI was not an independent variable in analyses, the differences between obese and non-obese individuals in response to food cues suggests that, even in

the non-obese range, BMI may play a role in response (Thomas, Doshi, Crosby, & Lowe, 2011).

When studies have manipulated food deprivation in food cue studies, they often have found significant differences in the way individuals react when hungry and full (Coletta et al., 2009; Demos et al., 2011; Ely et al., 2013; Nijs, Muris, et al., 2010). Therefore, the present study included two ERP recording sessions: one in a hungry state, and one following a meal. It also included questions probing for hunger level and time since last meal to ensure that this manipulation was successful.

Another factor that has varied greatly between studies is the content of stimuli presented. Most have compared food with high hedonic value to non-food objects (Babiloni et al., 2009; Blechert et al., 2010; Cornier et al., 2013; Demos et al., 2011; Kemmotsu & Murphy, 2006; Nijs et al., 2008; Nijs, Muris, et al., 2010; Watson & Garvey, 2013), while only a few (Coletta et al., 2009; Ely et al., 2013) have compared foods with high and moderate hedonic value. Additionally, some studies presented food cues with the instruction that this food will later be consumed (Burger & Stice, 2011; Stice, Yokum, et al., 2011), while others are simply presented with no expectation of actual consumption. Almost all studies used visual food cues, but one used food and non-food odors rather than images (Kemmotsu & Murphy, 2006). Because consumption of palatable, high calorie foods is particularly problematic for weight gain, the present study compared foods with high hedonic value to those with moderate hedonic value in order to gather information about whether HDs respond differently to these hedonically pleasing, diet-inconsistent foods differently than they do to healthier food images with lower hedonic ratings. This comparison was designed to provide more detailed information

about the role of the obesogenic food environment on brain processing and weight gain proneness than a comparison with non-food objects would.

In addition to the content of stimuli presented, the behavioral task performed affects brain response. Studies have also varied greatly on what participants were instructed to do while presented with food cues. Some were asked to passively view images (Coletta et al., 2009; Ely et al., 2013; Nijs et al., 2008), while some were given a minor task to ensure they were paying attention (e.g. identify which image has a person in it, imagine eating the food, count number of images in each category) (Blechert et al., 2010; Demos et al., 2011; Nijs, Muris, et al., 2010). Some were given more cognitively taxing tasks such as Go/No-go, Stroop, or an oddball paradigm (Babiloni et al., 2009; Nijs, Franken, et al., 2010; Watson & Garvey, 2013), while others were presented with a food image as a cue that they would be consuming it soon (Burger & Stice, 2011; Stice, Yokum, et al., 2011). While each of these techniques has its own benefits and downsides, the present study presented images with a minor task involved both to ensure attention and to gain information about the individual's dietary preferences for personalized stimulus categorizations. Participants were asked to rate each food image as "delicious" or "not delicious." While food items were chosen based on what is most commonly considered to be have high hedonic value (e.g. cake, pizza) and moderate hedonic value (e.g. cauliflower, plain rice), the participants' own ratings of whether or not they consider the food delicious were used in analyses to categorize foods into "delicious" and "not delicious" groups.

Finally, early (N2/EPN) and late (P3 and LPP) ERP components were measured as the dependent variables in this study, along with even earlier (C1/P1/N1) components

in an exploratory manner. ERP was chosen due to its high temporal resolution, which allows for the analysis of how the brain responds to food cues over time. Due to the difficulty in disentangling the N2 and EPN components, one early dependent variable during the time frame in which both of these occur, in order to capture effects in either of these components, was measured, and referred to as the N2. As P3 and LPP components do not overlap in time frame, they were each be targeted as a separate outcome variable. Prior studies have found these three components to be particularly relevant when comparing those with tendencies towards higher weight to those without a weight gain predisposition on response to food cues (Babiloni et al., 2009; Blechert et al., 2010; Kemmotsu & Murphy, 2006; Nijs, Franken, et al., 2010; Nijs, Muris, et al., 2010; Watson & Garvey, 2013). By focusing on both early and late components, the question of whether HD may be "down-regulating" their attention towards palatable foods was easier to examine, since higher-level executive functions such as inhibition take longer to put into effect than do initial visual perceptual processes (Woodman, 2010).

In summary, the present study compared ERP response to food cues in HDs and NDs. All participants were tested once when hungry and once when full, and viewed a selection of food images with either high or moderate hedonic value. They rated each image as either "delicious" or "not delicious" by clicking the left or right buttons on a computer mouse. ERP components of interest included an early latency component (N2/EPN), P3, and LPP.

### 1.7. Aims and Hypotheses

A model was analyzed including a three-way interaction between deliciousness (delicious, not delicious), dieting history (HD, ND), and hunger level (hungry, full).

Topographical factors (anterior-posterior; left-right-medial) were included in the model in order to examine differences in topographical expression of the hypothesized components. In order to understand this complex interaction, hypotheses refer to simpler two-way interactions.

1. To examine whether the role of hunger level on response to foods considered delicious differs between HD and ND.
    a. *Hypothesis 1*: The early (i.e. 200-300ms) components (N2/EPN) in response to delicious foods will be larger for NDs when hungry than when full. HDs, on the other hand, will not have an attenuation of early response to delicious foods when full. Their early components will be large both when hungry and when full.
    b. *Hypothesis 2*: Late components (P3 and LPP) in response to delicious foods will be smaller when full than when hungry in NDs. Late components will be similar in magnitude in both full and hungry states in HDs.
2. To examine whether the role of hunger level on response to foods not considered delicious differs between HDs and NDs.
    a. *Hypothesis 3*: Early and late responses to not delicious foods will be smaller when full than when hungry in both HDs and NDs.

# Chapter 2: Method

## 2.1. Participants

### 2.1.1. Recruitment

Participants aged 18 to 30 were recruited for this study from Drexel's student population as well as the surrounding Philadelphia community. Participants were recruited through flyers on campus and around Philadelphia, announcements in classes, and through Sona Systems, a website that allows Drexel undergraduates to participate in psychology research at the university. The study was described as recording the brain's responses to viewing images of food. In order to have sufficient power to compare HDs to NDs and to ensure a wide range of weights, we oversampled individuals in the overweight range and those with a history of dieting.

### 2.1.2. Inclusion/Exclusion criteria

In order to be eligible for the study, participants must have been right handed and have a body mass index (BMI) between 20kg/$m^2$ and 30kg/$m^2$. As the goal was to study factors that might contribute to weight gain and eventual obesity, only non-obese individuals were recruited. Exclusion criteria included use of a medication that affects body weight, energy expenditure, or brain function; presence of a medical or psychiatric condition that may have limited one's ability to comply with the study procedure; history of an eating disorder (Anorexia Nervosa, Bulimia Nervosa, or Binge Eating Disorder); being pregnant or planning to become pregnant in the next year; current dieting to lose weight; and being unable to consent.

**2.2. Procedure**

Interested potential participants were directed to the study website, where they read a brief description of the study, which provided sufficient detail for them to decide if they were still interested and would like to proceed with the screening process. They were asked to complete a 5-minute online screening questionnaire via Qualtrics, a secure online survey tool. Here they entered information about the inclusion/exclusion criteria listed above. Only after completing the questionnaire and being deemed eligible were participants asked to provide their name, email address, and phone number. Study coordinators then contacted eligible participants by email to schedule an in-person screening visit.

At the start of the in-person screening visit, informed consent for the visit was obtained from participants. The study coordinator then measured participant's height and weight, and the participant completed a series of screening questionnaires, including (1) a demographic sheet, (2) a basic health-related information sheet (including information on smoking status and current medications), (3) a questionnaire assessing diet and weight history, (4) a questionnaire assessing food preferences, (5) Restraint Scale, and (6) Positive and Negative Affect Scale (PANAS) (Trait). More detail is provided about these measures below. The study coordinator reviewed information to determine eligibility for the EEG portion of the study. Regardless of eligibility, all participants were compensated $10 for participation in the in-person screening portion of the study.

If eligible, the participant was randomly assigned to visit order (hungry first or full first). The two EEG visits were scheduled and instructions regarding visit preparation were given. Specifically, for the hungry visit participants were asked to only consume

water starting six hours prior to the visit. For the full visit, they were asked to eat a meal consisting of 400-600 calories one hour before the visit. Participants were asked to abstain from tobacco, alcohol, or recreational drug use for 24 hours prior to each visit. They were reminded to follow instructions 24 hours prior to each visit.

At the first EEG visit informed consent for the EEG portion of the study was obtained. The EEG procedure was the same for each visit. Skin on the forehead and behind the ears was cleaned with rubbing alcohol. Then the EEG cap was placed on the head and all electrodes were filled with saline gel. Impedance was tested, and was decreased to below 10 kOhm prior to data collection. Baseline EEG recordings were recorded for ten minutes (not used for the present study). Next, participants completed a short questionnaire assessing hunger level. Then they completed the food rating task. It was programmed with E-Studio 2.0 and displayed on an 18” monitor with a resolution of 640x480 pixels. Eighty food images were used in the paradigm. Half were chosen to have high hedonic value and half were chosen to have moderate hedonic value, based on ratings by a separate sample prior to the study. All stimuli were closely matched based on size, angle, and brightness. Each image included the food item on a plate that was 16cm in diameter. At a viewing distance of approximately 50cm, each picture occupied approximately 18 degrees of the visual angle horizontally and vertically. Examples of foods with high and moderate hedonic value can be seen in Figure 1. The stimulus order was randomized and the same order was shown to each participant. The same images were shown at each visit, but were presented in their mirror image form at the second visit, and in a different, also randomized, order from the first visit. Participants viewed a fixation cross for 750ms, followed by a larger cross shown for 500ms, alerting them that

the picture was about to be shown. The image was then presented for 2000ms, after which “Respond now” flashed for 250ms. Participants were instructed to click the left mouse button if they found the food delicious, and the right mouse button if they did not find the food delicious. A fixation cross remained on the screen for up to 2500ms, or until a response was given. Between each trial “OK to blink” was presented on the screen for several seconds. Prior to the experimental trials, 16 practice trials were shown.

Following the EEG recording on the first visit, the study coordinator did a semi-structured interview with the participant asking about any loss of control over eating during the past three months. Then another series of questionnaires was completed, including (1) Morningness-Eveningness Scale, (2) Pittsburgh Sleep Quality Index, (3) Eating Inventory, (4) Power of Food Scale (PFS), (5) Behavioral Inhibition and Activation Scale, (6) PANAS (State), (7) Body Image Assessment for Obesity, and (8) Menstrual Status, Medication, and Sleep Questionnaire. After the second visit, they again completed only the PFS, PANAS (State), and Menstrual Status, Medication, and Sleep Questionnaire.

After completion of the study, participants were compensated their choice of $30 or 4 psychology extra credit points plus $10.

**2.3. Measures**

Only the measures specifically related to the analyses of the present study will be reviewed in detail.

*Diet and Weight History Questionnaire* (Witt, Katterman, & Lowe, 2013). The DWHQ is designed to gain a detailed history of one’s attempts at dieting and weight fluctuations. Information about current dieting status and number of past diets was used

to categorize participants as “historic dieters,” “never dieters,” or ineligible. Additionally, the DWHQ measures weight suppression (WS), the difference between current weight and highest weight, which has been found to have adaptive and maladaptive consequences in non-clinical populations, but primarily maladaptive consequences in clinical populations, specifically individuals with AN and BN (Witt et al., 2013). WS is one marker of how successful one’s diet has been.

*Hunger Assessment* (Friedman, Ulrich, & Mattes, 1999). Participants were asked to self-report current hunger level, desire to eat, fullness, and the amount they currently felt able to eat on a 9-point scale at both EEG visits. This measure was used to ensure the experimental manipulation of hunger state had its intended effect.

*Body Mass Index*. BMI was assessed at the screening visit. Weight (in pounds) and height (in inches) was measured twice with shoes removed, using a calibrated digital scale with an attached stadiometer. Weight and height averages were calculated from the two measurements, and BMI was calculated using the average values. BMI was used to determine eligibility.

**2.4. Electrophysiological recording**

The electroencephalogram was recorded from an array of 25 electrodes mounted in an elastic cap that was interfaced to a DBPA-1 amplifier/digitizer (Sensorium Inc., Charlotte, VT). Vertical and horizontal eye movements were recorded from two electrodes placed on the face next to and below the left eye. During recording, all electrodes were referenced to electrode Cz. EEGLAB (Swartz Center for Computational Neuroscience) was used to clean EEG data. The EEG was sampled at 256 Hz. Data were re-referenced to an average reference calculated from all electrodes. Bad channels were

removed through visual inspection and interpolated from surrounding electrodes. Data were filtered using a 0.1 Hz high pass and 30 Hz low pass FIR filter. Next, responses to stimuli were reviewed to ascertain that sufficient “delicious” and “not delicious” responses were given. If fewer than seven responses were given to either category in a session, that session was not included in analyses because of insufficient data to create a valid ERP. In order to identify and remove artifacts associated with blinks and movements, gross artifact rejection was next run to remove entire epochs that were unusable for analysis. The range from 200ms prior to through 1000ms following stimulus presentation was scanned. If more than three channels contained peak amplitudes larger than 35Mv, that trial was rejected and was not included in further analyses. If between one and three channels contained peak amplitudes larger than 35Mv, these channels were interpolated from surrounding electrodes.

In order to calculate ERPs, the mean 200ms prestimulus served as baseline. ERPs were be averaged separately for HD and ND in hungry and full conditions, and then subdivided further into trials associated with a “delicious” response and a “not delicious” response, to investigate the moderating effect of palatability. Seven epochs were calculated: 50-90ms (C1), 80-110ms (P1 and anterior N1), 140-180ms (posterior N1), 150-220ms (anterior N2), 220-360ms (posterior N2), 300-550ms (P3), and 500-800ms (LPP).

**2.5. Data analysis**

SPSS v.22 was used to analyze the data. An Independent Samples t-test was used to compare hunger level in full and hungry states, to determine whether the hunger manipulation was successful. Scores on the Hunger Assessment were compared, as well

as mean hours since last meal eaten between conditions. Dieting groups were compared on demographic and weight-related variables.

Mean amplitudes in three temporal windows chosen based on those used in several previous experiments were used to quantify the ERPs: 180-300ms for the early component (reflecting N2 and EPN), 300-550ms for the P3 component, and 500-800ms for the LPP component (Hill et al., 2013; Nijs, Franken, et al., 2010; Schupp et al., 2007; Watson & Garvey, 2013). However, after reviewing the ERP waveforms, it was evident that the predefined N2 was capturing several waveforms and was not aligning accurately with the N2. Therefore, this component was redefined as 150-220ms in the anterior region and 220-360ms in the posterior region. Additionally, from visual inspection of the waveforms differences between conditions prior to 150ms were evident. Therefore, three additional epochs, the C1, P1, and N1, which have been found in previous research to reflect early visual attention, were added to analyses in an exploratory manner. Based both on previous research and visual inspection of the waves, the C1 epoch was defined as 50-90ms, the P1 as 80-110ms, and the N1 as 80-110ms in the anterior region and 140-180ms in the posterior region (Ernst et al., 2013).

Mixed factorial analyses of variance (ANOVAs) with within-subject factors of hunger (hungry, full), stimulus rating (delicious, not delicious) as well as hemisphere and anterior-posterior electrode position, and a between subject factor of dieting history (HD, ND), were used to analyze the ERP data. Specifically for the topographical factors, five electrode sites in the left hemisphere (FP1, F3, C3, P3, O1), middle (FPz, Fz, Cz, Pz, Oz), and right hemisphere (FP2, F4, C4, P4, O2) were chosen for analyses. Midline and lateral electrodes were analyzed separately. This provided ANOVA factors of hemisphere (left,

right) and anterior-posterior position (five levels) for the lateral electrodes, and anterior-posterior position for midline electrodes (Grainger, Lopez, Eddy, Dufau, & Holcomb, 2012). Visit order was also included as a between-subjects factor, and if it significantly interacted with hunger state, it was included as a covariate for all further analyses of that epoch. For all statistical analyses a Hunyh-Feldt correction was used for all repeated measures factors with greater than 2 degrees of freedom in the numerator. Significant and marginally significant interactions were decomposed within the region of interest for that component in order to better understand the pattern of activity seen. For the C1 and P1, this was the posterior region (O1, Oz, and O2, Luck, 2005). The N1 and N2 are typically maximal at both anterior (FP1, FPz, and FP2) and posterior (O1, Oz, and O2) regions at different times, so they were analyzed in both regions (Luck, 2005). The P3 and LPP have both been shown to occur maximally at parietal sites (P3, Pz, and P4; Luck, 2005; Olofsson et al., 2008)[1].

## Chapter 3: Results

### 3.1. Self-report measures and demographics

652 interested individuals completed our online screening questionnaire. Of these, 122 were eligible for and attended an in-person screening visit. Seventy-six were eligible

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[1] In the thesis proposal, it was stated that analyses would be run without participants using a medication that may affect brain activity, or who used alcohol, tobacco, or recreational drugs in the past 24 hours, in order to determine if they should be included in final analyses. This will be done in future analyses for publication. Additionally, it was stated that the relationship of BMI and WS with outcomes would be tested, and that if they were significant, they would be included as covariates. Because of the large number of analyses already done, these were not done for the thesis. Finally, z-score transformations of ERPs were not done for the thesis because the primary outcomes were related to the between-subjects effect of dieting history. They also may be incorporated in future analyses.

and invited to participate in the EEG portion of the study. Nine dropped out before completing both EEG sessions, leaving 67 participants who completed the entire study. Two participants were excluded from analyses due to problems with their data collection.

The final dataset included 65 participants. The sample was primarily white (50.0%) and Asian (28.1%), with 12.5% of participants identifying as Hispanic/Latino and 4.7% identifying as black. Thirty reported a history of past dieting, and 35 had never been on a diet to control their weight. HDs, relative to NDs, were similar in BMI ($t(63) = -0.13$, $p = 0.90$), younger ($t(63) = 2.23$, $p = 0.03$), and more weight suppressed ($t(39.59) = -1.92$, $p = 0.06$). Descriptive information by group can be found in Table 1.

*Hunger manipulation*. The hunger manipulation was successful. Participants rated themselves as significantly higher on all questions of the VMH when fasting than when full. For example, on a 1 – 7 scale where 1 = "nothing" and 7 = "a large amount," in response to the question "how much food do you think you could eat right now?" participants gave a mean rating of 2.22 (SD = 1.37) in the fed condition and 6.41 (SD = 1.86) in the fasting condition. Additionally, in a subset of participants for which the information was collected (time since last meal was not asked for the first several months of data collection; n = 47), they reported a mean of 9.61 (SD = 4.04) hours since their last meal in the fasting condition and 0.92 (SD = 0.42) hours in the fed condition, $t(46) = -14.01$, $p < 0.001$. Hunger ratings and hours since last meal in each condition did not significantly differ between dieting groups.

*Stimulus ratings*. On average participants rated 56.51% of the images as "delicious," 41.81% as "not delicious," and did not respond to 1.68% of images. However, there was wide variability in the way they split their responses. Out of the 80

stimuli presented on each visit, the number of “not delicious” responses ranged from 1 to 72, and the number of “delicious” responses ranged from 7 to 76. 55% of visits included at least one trial where no response was given. Of these trials, the mean number of missed trials was 2.45 (SD = 2.69, range: 1 – 16). Four sessions were excluded from analyses because the number of either “delicious” or “not delicious” responses was below six, thus providing insufficient trials to calculate the average ERP.

*Visit order effects*. Although condition order was randomized to avoid systematic effects of visit order on results, an interaction between visit number and hunger condition was tested on each ERP epoch. The interaction was significant for the 50-90ms (C1), 220-360ms (posterior N2), 300-550ms (P3), and 500-800ms (LPP) epochs, and therefore order of fasted or fed (dummy coded as 0 or 1) was included as a covariate in all analyses for those epochs.

**3.2 ERP**

*C1 (50-90ms).* Controlling for visit order effects, a C1 dieting history*anterior-posterior electrode location effect was found (Lateral: $F(1.40,72.26) = 4.16$, $p = 0.032$; Midline: $F(1.56,81.10) = 3.37$, $p = 0.051$). As the C1 is mainly seen in posterior electrodes, reflecting activity in the primary visual cortex, further analyses examined the main effect of dieting history on amplitude in posterior electrodes (O1, Oz, and O2). A significantly larger C1 was seen in ND than HD groups ($F(1,62) = 7.62$, $p = 0.008$; M = 2.75 (SD = 2.42) and 0.98 (SD = 2.65) respectively; see Figures 2, 4, and 7).

*P1 (80-110ms).* A significant three-way interaction between hunger, dieting history, and anterior-posterior electrode location was found on P1 amplitude (Lateral: $F(2.11,113.99) = 4.07$, $p = 0.018$; Midline: $F(2.54,137.05) = 3.71$, $p = 0.018$). In order to

examine the relationship between hunger and dieting history in the occipital region, where the P1 effect is most pronounced, a post-hoc ANOVA was run to examine the two-way hunger*dieting history interaction within posterior electrodes (O1, Oz, and O2). The interaction was marginally significant, $F(1,54) = 3.55$, $p=0.065$ . Analysis of simple effects revealed that, in the ND group, a smaller P1 effect was seen when hungry ($M = 2.06$, $SD = 0.94$) than when full ($M = 2.98$, $SD = 0.95$; $F(1,54) = 3.55$, $p = 0.018$). No simple effect of hunger was seen in the HD group (hungry: $M = 2.31$, $SD = 0.90$; full: $M = 2.44$, $SD = 0.91$), and no simple effects of dieting history were found in either hunger state (see Figures 2, 4, and 8).

Additionally, in midline electrodes a marginally significant 4-way, hunger*dieting history*stimulus type*anterior-posterior electrode location interaction was found ($F(1.97,106.16) = 2.70$, $p = 0.073$). To follow up, the hunger*dieting*stimulus type interaction was examined in electrode Oz, since the P1 is seen in posterior regions. The interaction was marginally significant ($F(1,54) = 2.85$, $p = 0.097$). It was then broken down further, with the hunger*dieting history interaction examined for delicious and not delicious stimuli separately. For not delicious stimuli, the interaction was significant, $F(1,54) = 7.90$, $p = 0.007$. Again, a simple effect of hunger within NDs was found, $F(1,54) = 19.20$, $p < 0.001$, with a smaller P1 seen in the hungry state ($M = -0.38$, $SD = 1.02$) than the full state ($M = 1.23$, $SD = 1.01$). P1 amplitude was not significantly affected by hunger among HDs, and no simple effects of dieting history were seen within hunger groups. The hunger*dieting history interaction was not significant for delicious stimuli, $F(1,54) = 0.62$, $p = 0.978$.

*N1 (anterior: 80-110ms; posterior: 140-180ms).* Because the anterior N1 occurred during the same time frame as the posterior P1, the hunger*dieting history*anterior-posterior electrode location interaction referenced in P1 analyses was examined as an N1 effect by testing the hunger*dieting history interaction in anterior (FP1, FPz, FP2) electrodes and was found to be marginally significant ($F(1,54) = 3.30$, $p = 0.075$). There was a marginally significant simple effect of hunger among NDs, $F(1,54) = 3.68$, $p = 0.060$, with a larger N1 seen in the full ($M = -1.48$, $SD = 0.44$) than hungry ($M = -1.06$, $SD = 0.46$) state. N1 did not significantly differ by hunger state among HDs (full: $M = -0.94$, $SD = 0.43$; hungry: $M = -1.07$, $SD = 0.44$), and no simple effects of dieting history were seen within hunger states (see Figures 3 and 4).

The midline 4-way hunger*dieting history*stimulus* anterior-posterior electrode location interaction referenced in P1 analyses was also tested in electrode FPz to determine whether hunger*dieting history*stimulus interacted on the anterior N1 effect. The interaction was marginally significant, $F(1,54) = 2.84$, $p = 0.098$. As was done to test the direction of the interaction on the P1, hunger*dieting history was tested within delicious and not delicious stimuli separately. The interaction was significant for not delicious stimuli, $F(1,54) = 4.08$, $p = 0.035$, with a simple effect of hunger seen for NDs only, $F(1,54) = 4.08$, $p = 0.048$. NDs had a larger N1 when full ($M = -1.11$, $SD = 0.49$) than when hungry ($M = -0.42$, $SD = 0.51$) in response to not delicious stimuli. The response of HDs did not significantly differ by hunger state (full: $M = -0.28$, $SD = 0.48$; hungry: $M = -0.62$, $SD = 0.49$). The hunger*dieting history interaction was not significant for delicious stimuli, $F(1,54) = 0.29$, $p = 0.60$ (see Figure 9).

*N2 (anterior: 150-220ms; posterior: 220-360ms).* A hunger*dieting history interaction was marginally significant on mean amplitude from 150-220ms in lateral electrodes, $F(1,53) = 3.91$, $p = 0.053$. In order to test whether this represented an anterior N2 effect, the interaction was tested in lateral anterior electrodes (FP1 and FP2). The interaction was not significant in the anterior region, $F(1,53) = 0.03$, $p = 0.867$. The hunger*dieting history interaction was also marginally significant on mean amplitude from 220-360ms in lateral electrodes, controlling for visit order effects, $F(1,52) = 3.77$, $p = 0.058$. As the N2 was seen in the posterior region during this time frame, the interaction was tested in electrodes O1, Oz, and O2. It was not significant in the posterior region, $F(1,52) = 1.51$, $p = 0.225$. A main effect of hunger, but not dieting history, was found, $F(1,52) = 8.40$, $p = 0.005$. A larger N2 mean amplitude was found when hungry ($M = 1.82$, $SD = 0.29$) than when full ($M = 2.03$, $SD = 0.33$). A marginally significant three-way stimulus*dieting*anterior-posterior electrode location interaction was also found on mean amplitude in the 220-360ms range, controlling for visit order effects (Lateral: $F(1.71,88.94) = 3.14$, $p = 0.054$; Midline: $F(1.97,102.26) = 3.31$, $p = 0.041$). However, when the stimulus*dieting interaction was tested in posterior (O1, Oz, O2) electrodes where the N2 is expected to be seen at this time range, the interaction was not significant ($F(1,62) = 1.35$, $p = 0.250$). These results suggest no significant effects of dieting history on N2 mean amplitude (see Figures 2, 3, and 6).

*P3 (300-550ms).* A hunger*stimulus*dieting interaction was marginally significant in lateral electrodes, controlling for visit order effects ($F(1,52) = 3.05$, $p = 0.087$). Because the P3 is maximally seen in parietal electrodes, this interaction was tested within electrodes P3 and P4. The interaction was not significant, $F(1,52) = 0.79$, p

= 0.377. However, a significant hunger*dieting status interaction was seen in parietal P3 amplitude, F(1,52) = 4.53, p = 0.038. Examination of simple effects found a marginally significant simple effect of hunger among the HD group, F(1,52) = 2.84, p = 0.098, with a larger P3 seen in the hungry state (M = 1.03, SD = 0.33) than full state (M = 0.69, SD = 0.33), but not within the ND group (hungry: M = 0.75, SD = 0.34; full: M = 1.02; SD = 0.34). No simple effects of dieting history were found within hunger states. No other P3 effects of dieting history were found (see Figures 5, 6, and 10).

*LPP (500-800ms).* A dieting history*hunger*anterior-posterior electrode location interaction was seen controlling for visit order effects (Lateral: F(1.86,96.48) = 3.61, p = 0.034; Midline: F(2.10, 109.35) = 3.78, p = 0.024). Because the LPP is seen most strongly in the parietal region, the dieting history*hunger interaction was tested in parietal electrodes (P3, P4, Pz). It was significant (F(1,52) = 9.74, p = 0.003), with simple effects of hunger for HDs and NDs in opposite directions. HDs had a larger LPP when hungry (M = 0.55, SD = 0.10) than when full (M = 0.34, SD = 0.12; F(1,52) = 5.07, p = 0.029), whereas NDs had a larger LPP when full (M = 0.55, SD = 0.12) than when hungry (M = 0.32, SD = 0.10; F(1,52) = 4.71, p = 0.035). Simple effects of dieting status within each hunger condition were not significant (see Figures 5, 6, and 11).

## Chapter 4: Discussion

The aim of the current study was to investigate the relationship between dieting history, hunger, and hedonic value of food on neural response to visual food cues. While existing research has found differences in food cue responsivity between individuals of different weights, as well as those demonstrating varying levels of cognitive restraint with respect to food, a history of dieting, which has repeatedly been shown to predict

future weight gain, had not previously been tested in this way. It was hypothesized that satiety would have a greater inhibitory effect on brain responses to food cues in NDs than HDs, when viewing delicious foods. Response to moderately palatable food cues was expected to decrease when full compared to when hungry in both NDs and HDs. Based on prior research, the described hunger by dieting history interaction was hypothesized on the N2 component, but a main effect of hunger was hypothesized on later P3 and LPP components. These hypotheses were, for the most part, not supported. No differences between HD and ND groups were found in N2 response to either type of food. A hunger by dieting history interaction was found on P3 amplitude in an unexpected direction; the HD group had a differential response based on hunger state, whereas the ND group did not, suggesting that HDs demonstrated more motivation towards food cues when hungry than when full, whereas NDs' attentional response was unaffected by hunger level. On even later LPP response, this interaction was again seen, with effects of hunger in both groups; the HD group had a larger LPP when hungry than when full, and the ND group had a larger LPP when full than when hungry, suggesting that hunger attenuated the sustained attentional response to food in NDs but magnified it in HDs. Neither the P3 nor LPP pattern was significantly different based on food palatability. Null findings have been found by others in regards to these epochs, though prior research has examined other diet-related measures besides historical dieting. For example, Watson and Garvey (2013) found no differences between restrained and unrestrained eaters in N2, P3, or LPP response to food compared to non-food stimuli in a go/no-go paradigm. As they did not control for hunger, results cannot be directly compared, but the lack of a dieting history by palatability interaction is consistent with their findings. Additionally, Blechert, et al.

(2010) found no differences in LPP amplitude between restrained and unrestrained eaters (as defined by the RRS) when passively viewing food and non-food images. The fact that in the present study the HD group had a larger P3 and LPP when hungry than when full, a difference not seen (or seen in the opposite direction) in the ND group, was unexpected. A history of dieting predicts future weight gain, suggesting an attraction to food beyond physiological need. If the increased P3 and LPP amplitudes do in fact reflect increased conscious attention towards the stimulus, these findings indicate that HDs may actually be more successfully down-regulating attention towards food when they are not physiologically hungry, compared to those who have not dieted, a pattern that has been seen in current dieters in the past (Lowe et al., 1991; Lowe, 1995). As BMI in this sample did not differ by dieting history, and the HD group was significantly more weight suppressed than the ND group, perhaps the sample selected was in fact a group of successful dieters. Although they did not report presently being on a weight loss diet, they may have been able to continue employing strategies to keep their weight below what it once was, at least when it comes to sustained, conscious attention towards food cues. Physiological hunger due to fasting, on the other hand, may be strong enough of a driver in HDs that they are no longer able to down-regulate their response to food, resulting in an equivalent, or even higher, P3/LPP compared to NDs in the hungry state. Another explanation for the findings could be the idea that weight gain prone individuals, prior to weight gain, respond in ways opposite to those who are already obese. For example, a very healthy metabolic profile has been associated with weight gain in Pima Indians, and an increased reward response to food receipt predicts weight gain and an eventual reduction in reward response to food receipt (Stice et al., 2009; Swinburn et al.,

1991). While Nijs, Muris, and colleagues (2010) found that overweight/obese participants showed a reduced P3 to food cues when hungry compared to normal weight individuals, our HDs showed a larger P3 response when hungry than when full, a difference not seen in NDs. Perhaps a blunted hungry P3 in response to food develops with weight gain, and those with an exaggerated hungry P3 at a nonobese weight are primed to gain weight over time. Importantly, when differences between dieting groups were found, they were contingent upon hunger state. While main effects of hunger were found across participants, no main effects of dieting history were seen. This pattern stresses the importance of controlling for, and ideally manipulating, hunger state when examining ERP response to food cues, something that has infrequently been done in past literature.

Most past studies that saw N2 effects involved tasks that required response inhibition (Kemmotsu & Murphy, 2006; Nijs, Franken, et al., 2010). Our task, on the other hand, only involved passive viewing of food cues and rating their palatability. The lack of cognitive inhibition required for our task may explain why differences between HDs and NDs were not seen in N2 response. Perhaps if they had been asked to do a task relating to food cues that involved response inhibition or other executive control processes, a difference in the way palatable foods are processed would have been revealed.

Unexpectedly, differences were seen in much earlier components than have previously been studied with respect to food cue responsivity. Because upon visual inspection of the ERP difference waves the C1, P1, and N1 appeared to be more responsive to variables of interest than the later, hypothesized components, they were analyzed in an exploratory manner.

Most surprising was the main effect of dieting history on C1 mean amplitude. The C1 is thought to be generated from the primary visual cortex, and respond to stimulus parameters in a serial, bottom-up process that analyzes increasingly complex information (Bar et al., 2006; Luck, 2005). However, more recent models suggest that top-down processes facilitate visual object recognition by rapidly perceiving a low spatial frequency version of the object to determine several possible items it could be, thus informing the slower, more deliberate bottom-up pathway (Bar et al., 2006). These top-down processes may have an effect on C1 amplitude (Zani & Proverbio, 2012). In our study because all participants were exposed to the same stimuli, C1 differences cannot be explained by visual parameters of the images. These results suggest that individuals with a history of dieting may perceive food stimuli differently than those who have not dieted, before they consciously even know they have seen an image[2].

The P1 is thought to reflect preconscious spatial attention, and is sensitive to state of arousal (Luck, 2005). While originally the P1 was assumed to be determined solely by physical characteristics of an image, more recent research has found amplifications of the P1 by both aversive and appetitive stimuli (Kuhr, Schomberg, Gruber, & Quirin, 2013; Olofsson et al., 2008). While the NDs showed an increased P1 amplitude to food cues when full compared to when hungry, no effect of hunger state was seen on P1 amplitude

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[2] A potential alternative explanation for these findings could be the fact that midway through the study, there was a change in the room where data were collected due to a department move. Additionally, 33.3% of participants run in one location were in the HD group, whereas 57.9% of participants run in the other location were in the HD group. Therefore, although all efforts were made to replicate the characteristics of the first room after the move, location could account for the C1 differences seen between dieting groups. However, dieting history and location did not significantly interact to affect C1 mean amplitude, decreasing the likelihood that effects are driven by data collection location.

among HDs. This pattern is more similar to hypotheses than that of the later components, in that NDs were more sensitive to hunger state than HDs were. However, the direction of findings, where NDs were paying more preconscious attention to food when they were full than when hungry, is surprising. This is the opposite of what Stockburger and colleagues (2008) found when comparing ERPs in a fasting and full state in response to palatable food cues. Overall their participants had larger early positive potentials when hungry than when full, although this was seen later (170-320ms) than the P1 effect in this study (80-110ms).

The N1 pattern of activity seen mirrored that of the P1. That is, at a marginally significant level, HDs were unaffected by hunger state in their N1 mean amplitude, where NDs had a larger N1 when full than when hungry, particularly in response to not delicious foods. N1 is thought to reflect stimulus classification as an early mechanism to determine which stimuli require further processing (Luck, 2005). This pattern would indicate that, while cognitive effort given to classify foods does not depend on hunger level for HDs, NDs give more effort when they are full than when they're hungry. Perhaps NDs deem food cues worthy of further processing without much effort when they are hungry, as their physiological state is priming them to be sensitive to suggestions of consumption. When full, on the other hand, it might require more effort to categorize foods, especially ones they don't consider delicious, as worthy of further processing. According to the stimulus classification explanation, HDs gave equal attention to food cues regardless of hunger state. Their internal state did not determine the effect that external cues had on neural processing. These findings are in line with the idea that those who are obese, or perhaps on the path to future obesity, are more reliant on external than

internal cues to determine eating behavior (Rodin & Slochower, 1976). Other research has found the N1 to be larger for appetitive stimuli (dessert) than neutral stimuli (rock), particularly for those with higher trait levels of approach motivation as measured by the BIS/BAS (Carver & White, 1994; Gable & Harmon-Jones, 2010), as well as for erotic stimuli in comparison to other pleasant stimuli (Kuhr et al., 2013). Explaining the present findings according to these results would suggest that NDs find food cues more appetitive when they are full, rather than when they are hungry, whereas HDs find food cues equally appetitive regardless of hunger level.

Herman and Polivy's boundary model (1984) suggests that human biology regulates eating behavior by setting both a "hunger boundary" and a "satiety boundary." Between these boundaries exists a zone of biological indifference with no strong feelings of hunger or fullness. These authors suggest that restrained eaters assessed by the RRS, that is, individuals who report being highly conscious of what they eat and with large weight fluctuations, tend to override their biological hunger boundary in an attempt to eat less than desired. Over time this behavior leads to reduced sensitivity to the biological hunger and satiety signals, thus extending the zone of biological indifference in restrained eaters (Stroebe, van Koningsbruggen, Papies, & Aarts, 2013). The present study's findings of a lack of effect of hunger state on automatic, early neural response to food in HDs aligns with the boundary model. While NDs are sensitive to their biological hunger/satiety boundaries, responding differently to food cues depending on hunger state, the level of hunger and satiety induced by the manipulation in this study may have been too weak to reach HDs' extended hunger/satiety boundaries. If HDs are in their biological

zone of indifference in both conditions, it logically follows that their early neural response to food cues would not differ by condition.

On the whole, in both early and late time processing time frames, NDs tended to show a larger response to foods when full than when hungry. HDs, on the other hand, differed in their early and late responses to the hunger manipulation. In preconscious time frames HD responses to food were unaffected by hunger. In later time frames, on the other hand, a larger response was seen when hungry than when full.

The present study's comparison of foods rated as delicious and not delicious was designed to be an improvement upon the past trend of comparing highly caloric food to non-food items, in order to isolate a "hedonic effect" rather than measuring a more general "food effect." After 110ms after stimulus presentation, dieting history did not interact with stimulus rating; that is, HDs and NDs were similar in their differential processing of foods they deemed delicious and not delicious. The lack of significant findings suggests that differences seen in prior studies between groups in later, conscious processing of food with high hedonic value compared to non-food cues (Kemmotsu & Murphy, 2006; Nijs, Franken, et al., 2010; Nijs, Muris, et al., 2010) may have been the result of a general food effect, rather than the hedonic value of the food specifically. However, in our study, for both P1 and anterior N1 amplitude, a three-way interaction was seen between dieting history, hunger, and stimulus type. Breaking the interaction down revealed that the dieting history*hunger interaction was only significant for stimuli deemed not delicious. HDs did not respond differently based on hunger state to any type of food cue, whereas NDs responded more strongly when full than when hungry only to not delicious foods. Perhaps then hedonic value does matter for early, preconscious

processing; delicious foods may activate a hedonic network strong enough to override the biological hunger and satiety boundaries of even a non-dieter (Herman & Polivy, 1984; Lowe & Butryn, 2007), resulting in a lack of differential activation based on hunger for delicious foods for both HDs and NDs.

Because stimulus categories were determined by participant rating, “delicious” and “not delicious” items varied widely between participants. While using individual ratings assures that personal food preference is taken into account in categorization, it also means that groups cannot be standardized across individuals, that energy density is not necessarily different between delicious and not delicious groups, and that deliciousness findings may be different than previous studies that use standardized categorization methods (Nijs et al., 2008; Nijs, Muris, et al., 2010; Watson & Garvey, 2013). It would be interesting to examine whether a standardized hedonic value categorization based on energy density and mean ratings from a larger sample would elicit similar results. Perhaps objective characteristics of food have a more powerful effect on attentional processing than subjective ratings do, as subjective deliciousness ratings are likely to be affected by factors beyond taste preferences alone (e.g. assumptions about expected responses, food avoidance for reasons relating to weight, hunger, recently eaten foods).

Because a (marginally significant) three-way interaction between dieting history, hunger, and self-rated food deliciousness was found on P1 and N1 amplitude, perception of deliciousness appears to be processed already on some level by 110ms. Whether energy density and standard hedonic value determinations similarly come into play in such early object processing timeframes is unknown. The unique information provided in

this study about individual food ratings as well as the ability to categorize foods by energy density and mean hedonic value ratings provides an avenue to explore this question. Concordance of individual ratings with standardized categorization could also be compared between HDs and NDs, to see if HDs tend to rate foods as systematically more or less appealing than NDs. Emerging evidence suggests that during a behavioral weight loss program, reward-related activity towards healthy, low energy density foods can actually increase (Deckersbach et al., 2014). While the permanence of these changes after the program ends is unknown, it could be that HDs experience increased attraction towards healthy foods due to their past diets.

A particular challenge to interpreting ERP results is the lack of a basis on which to infer what is driving increased attention reflected by a larger ERP component. The present study compared dieting groups that have infrequently been studied in the past according to our categorization criteria. There is little basis on which to base interpretations of findings, especially for groups that by definition experience conflicting motivations when it comes to food cues (Stroebe et al., 2013), that is, an appetitive drive to consume food with high hedonic value, as well as motivation to avoid consumption to meet weight and dieting goals. From this framework, an increased ERP component could reflect increased motivation toward either of these goals, especially if the foods are not perceived to be "diet-consistent." Additionally, a smaller P3 or LPP component could reflect an innate lack of drive towards the stimulus, or, conversely, a conscious down-regulation towards the food cue in alignment with one's dieting goal. Much of the prior literature attempting to explain ERP response to food cues has struggled with the same

interpretive concern (Babiloni et al., 2009; Blechert et al., 2010; Kemmotsu & Murphy, 2006; Nijs, Muris, et al., 2010).

Strengths to the present study include a relatively large sample size, objective measures of ERP response to food cues in both the hungry and full state, and a novel classification of individuals based on dieting history, an empirically supported predictor of future weight gain. Several limitations to the current study exist. The findings related to very early visual processing (C1, P1, N1) were not hypothesized, as no previous work has examined response this early with respect to food cues. Therefore replication is necessary to interpret them with confidence. Additionally, stimuli were categorized based on participant rating. Results may have differed with a standard set of food cues that have been validated in terms of energy density and hedonic value (e.g. Food-pics; Blechert, Meule, Busch, & Ohla, 2014) and for which ratings were standardized across participants. Another limitation could be the relatively lenient criteria for HDs. It could be that individuals with only one past weight loss diet tend to respond to food cues differently than those with a long history of dieting and weight cycling, and thus by including them in the HD group, between-group differences were harder to see. Along these same lines, retrospective self-report was used to determine dieting history, which may be prone to errors in recall or inaccurate responses due to demand characteristics. Finally, as participants were primarily college students and were all female, results cannot be generalized to other populations with confidence.

In order to further examine the early processing differences between HDs and NDs, future studies should use both food and non-food objects in order to determine whether these effects are food-specific. It would also be interesting to categorize foods

based on standardized hedonic value rather than individual ratings to see if effects differ. Future research should also examine whether response to food cues measured with ERPs is a mediator between dieting history and future weight gain. If it is, ERPs may provide an avenue to better predict those most prone to weight gain based on their interaction with the food environment, thus informing obesity prevention programs.

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# Appendix A: Tables and Figures

Table 1. Participant weight and age information (Mean(SD)).

| | HD (n = 30) | ND (n = 35) | Total (N = 65) |
|---|---|---|---|
| BMI | 24.01 (2.70) | 23.93 (2.70) | 23.97 (2.68) |
| Weight | 140.05 (21.15) | 144.29 (20.47) | 142.7 (20.74) |
| WS | 7.53 (9.41) | 4.02 (3.97) | 5.70 (7.26) |
| Age | 21.04 (2.04) | 22.44 (2.93) | 21.77 (2.62) |

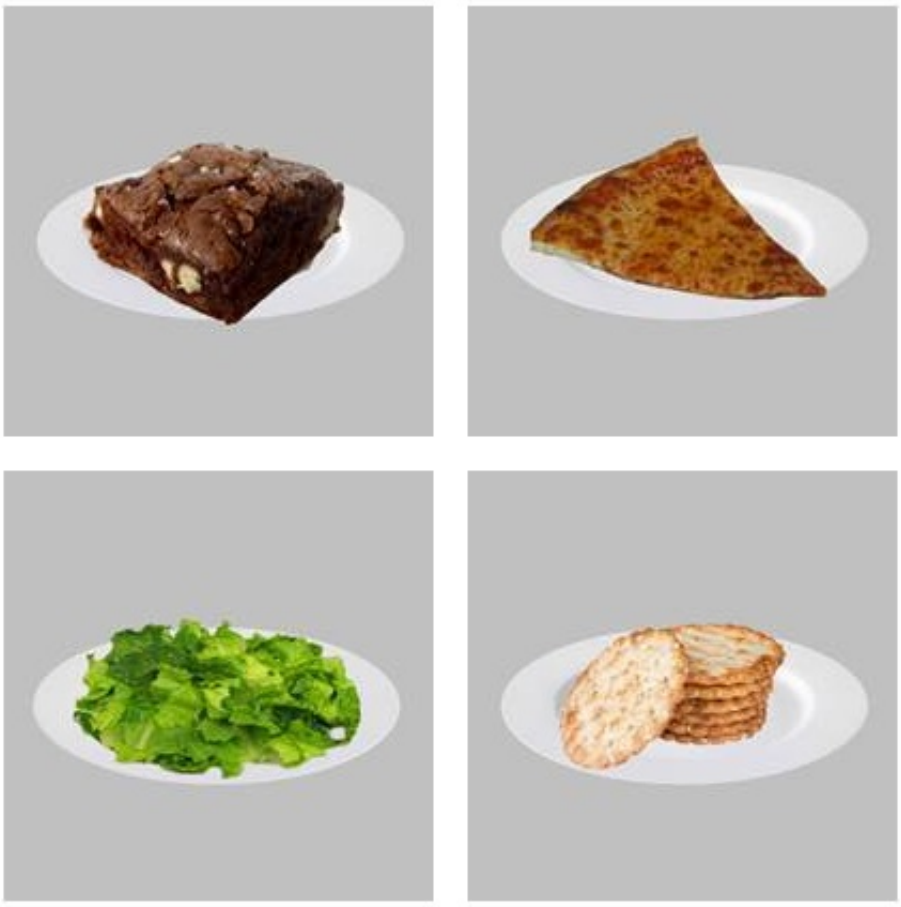

Figure 1. Examples of highly palatable (top) and moderately palatable (bottom) food stimuli.

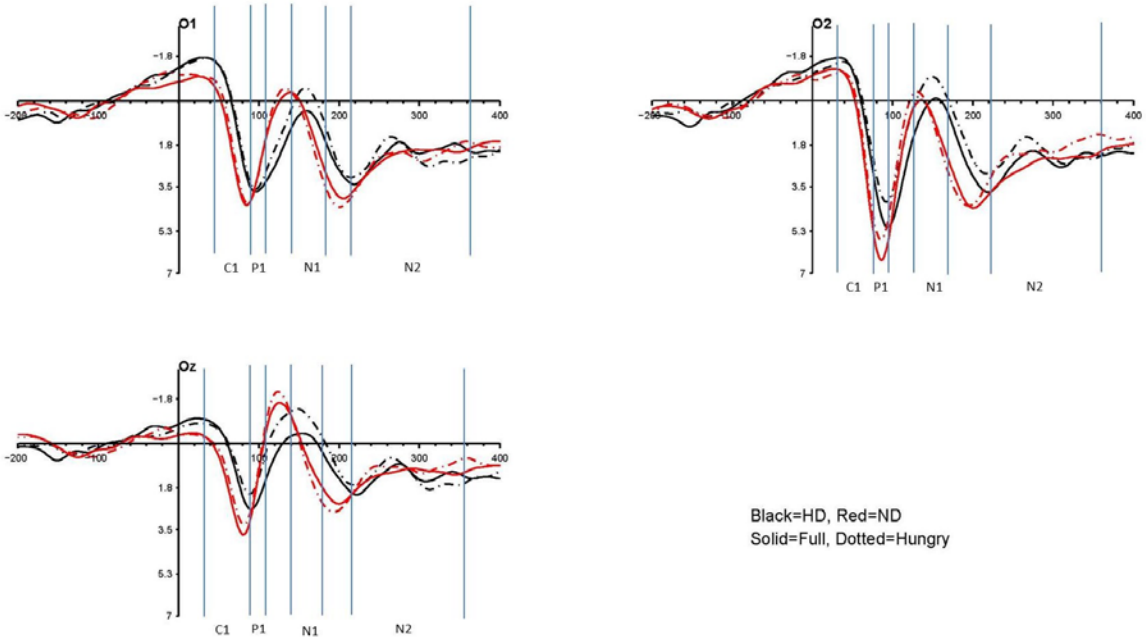


Figure 2. C1, P1, N1, and N2 epochs in posterior electrodes. Y-axis shown in mV and x-axis in ms.

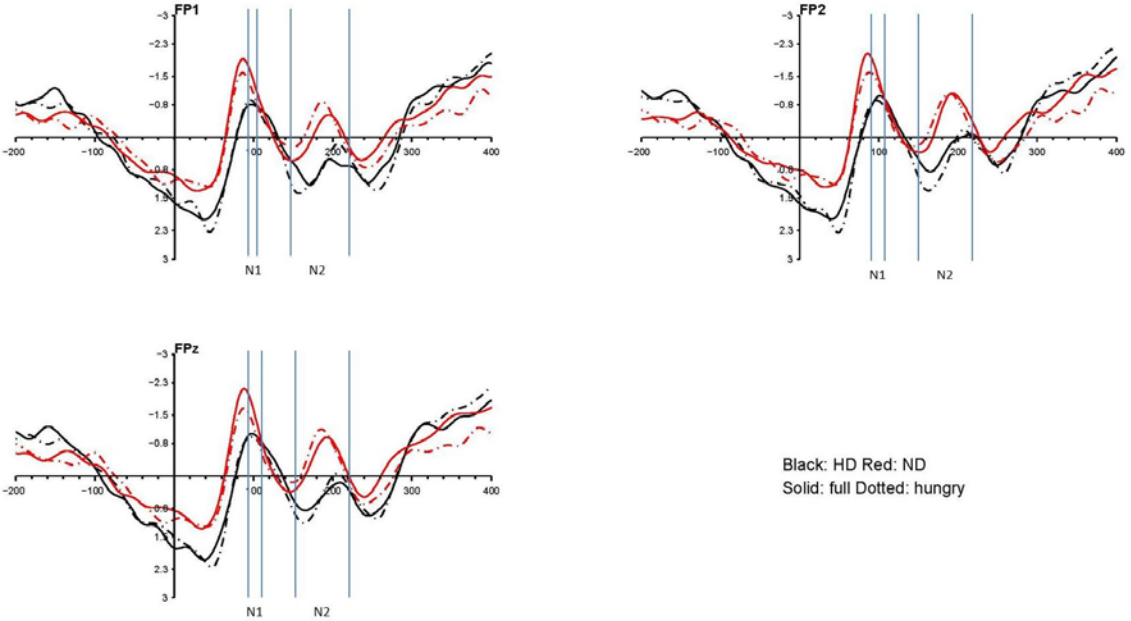


Figure 3. N1 and N2 epochs in anterior electrodes. Y-axis shown in mV and x-axis in ms

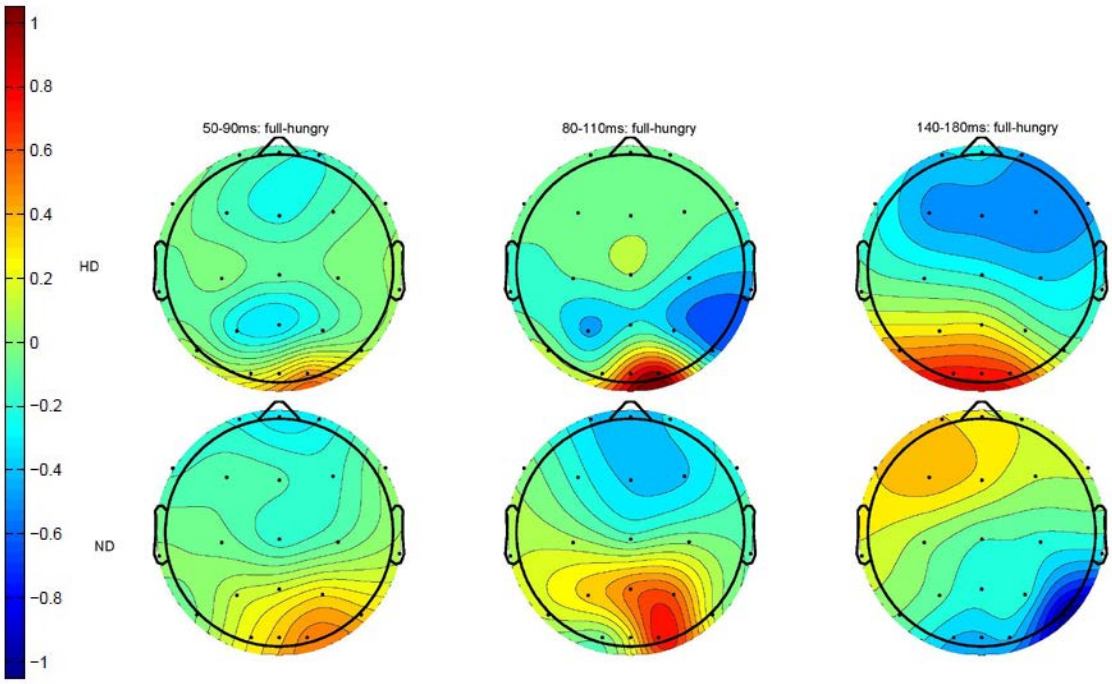


Figure 4. Scalp map of C1, P1 and N1 (anterior and posterior) epochs. Difference in mV between full and hungry states shown for HDs and NDs.

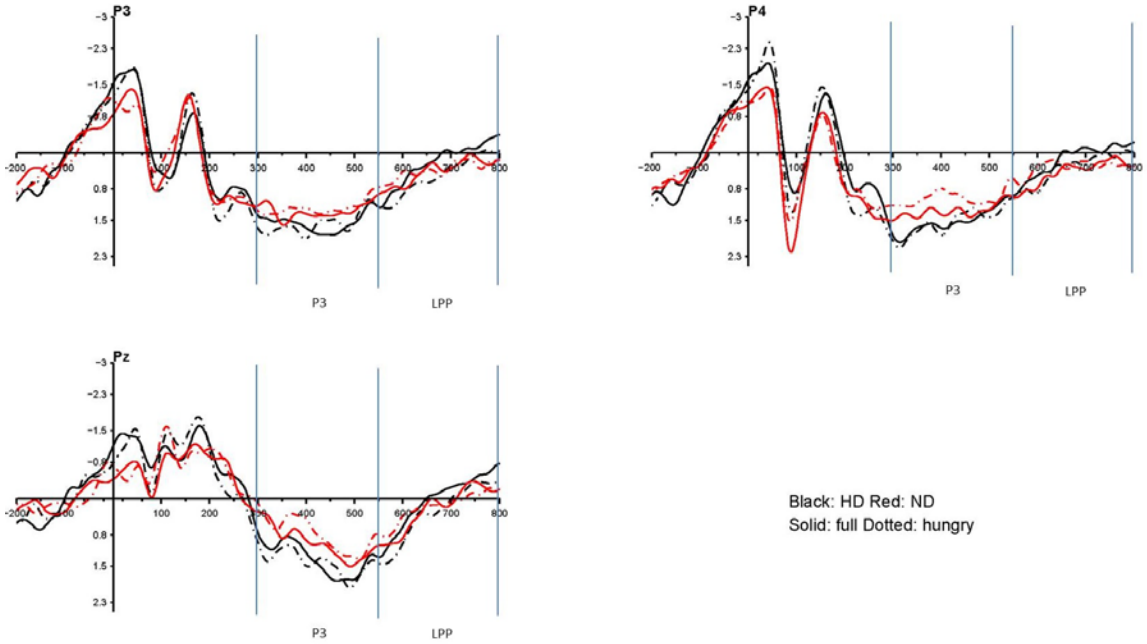


Figure 5. P3 and LPP epochs in parietal electrodes. Y-axis shown in mV and x-axis in ms.

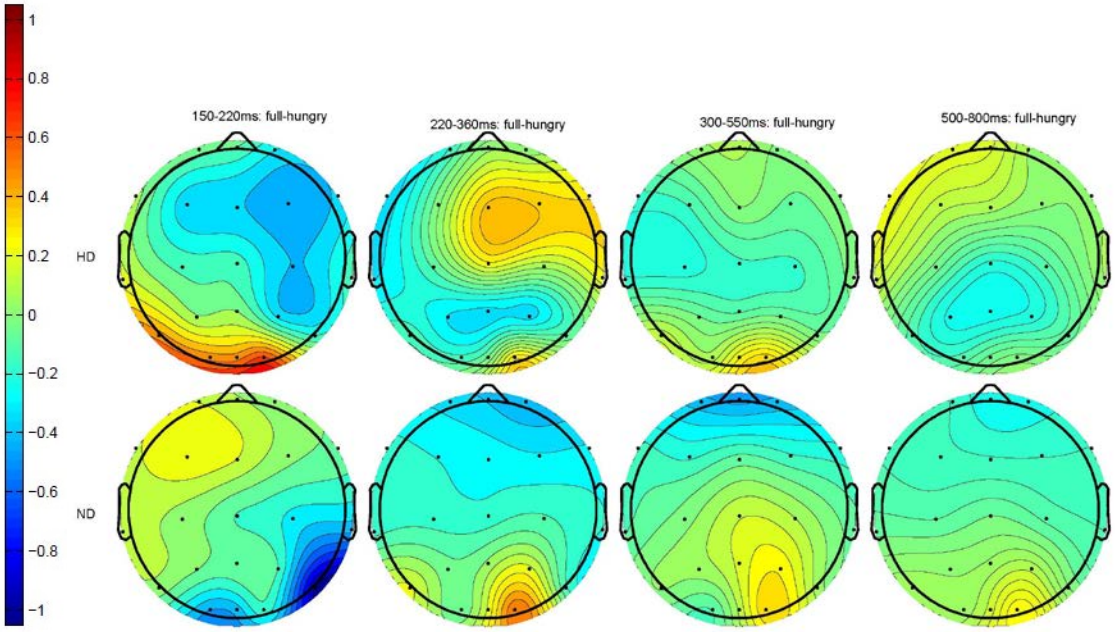


Figure 6. Scalp map of N2 (anterior and posterior), P3, and LPP epochs. Difference in mV between full and hungry states shown for HDs and NDs.

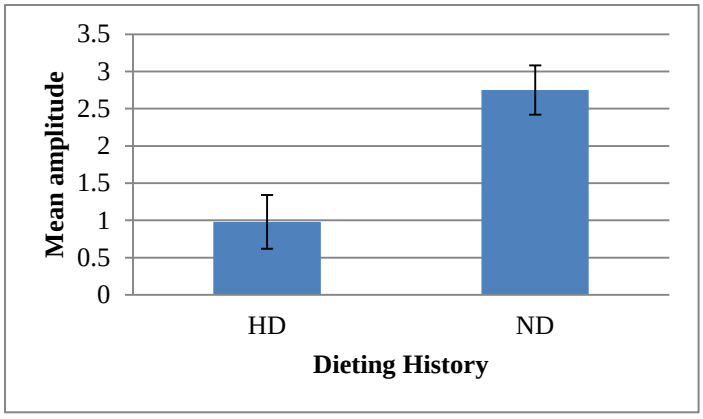


Figure 7. Main effect of dieting history on C1 mean amplitude, averaged across O1, Oz, and O2, $p = 0.008$.

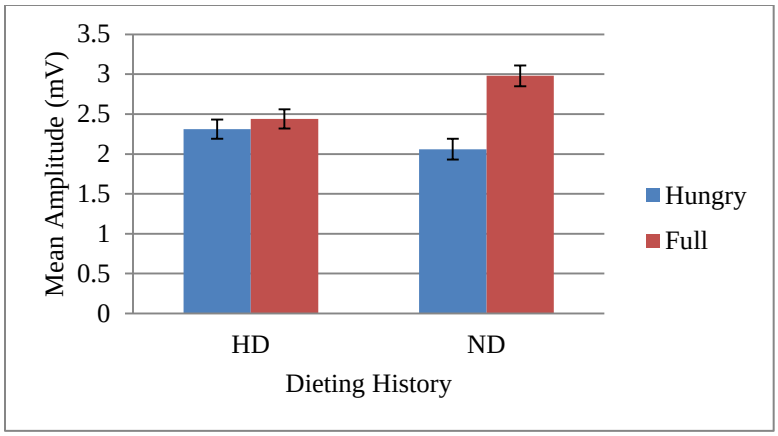


Figure 8. Dieting history by hunger state interaction on P1 mean amplitude, averaged across O1, Oz, and O2, $p = 0.065$.

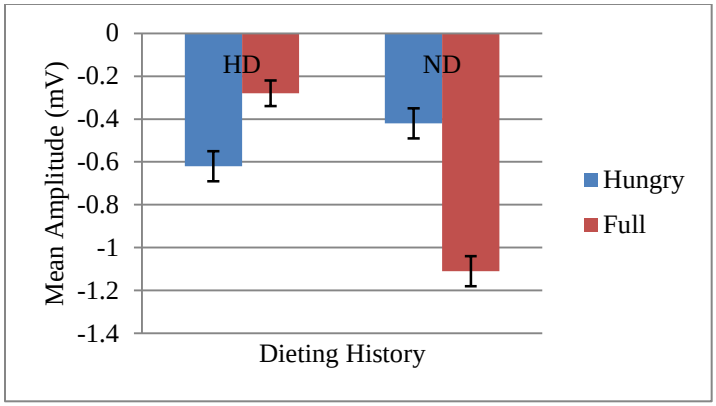


Figure 9. Dieting history by hunger state interaction on N1 mean amplitude, within "not delicious" stimuli, FPz, $p = 0.035$.

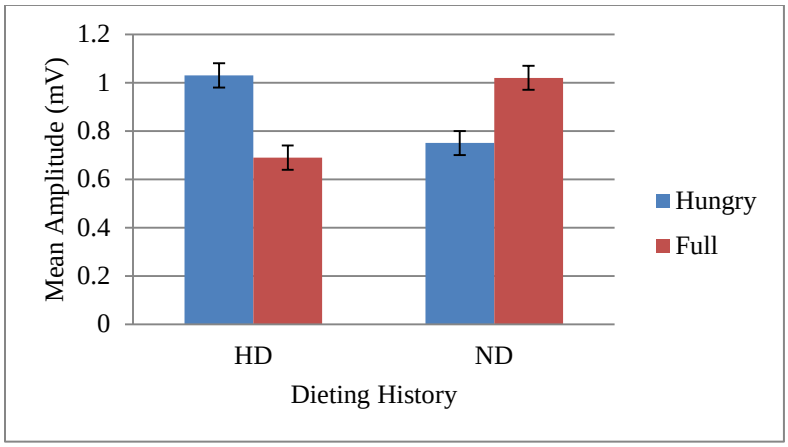


Figure 10. Dieting history by hunger interaction on P3 mean amplitude, across P3 and P4, $p = 0.038$.

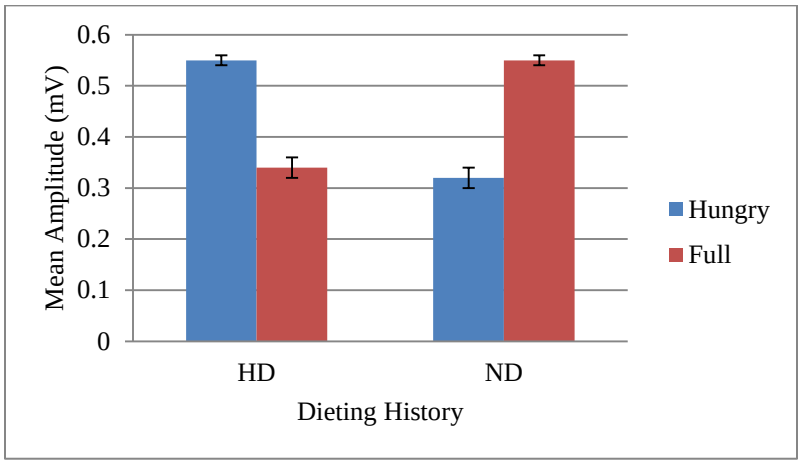


Figure 11. Dieting history by hunger interaction on LPP mean amplitude, across P3, Pz, and P4, $p = 0.008$.

## Appendix B: Self-Report Measures

Dieting and Weight History Questionnaire

DWHQ

1. What is the most you have ever weighed since reaching your current height? (Do not count any weight gains due to medical conditions or medications). The most I have weighed since reaching my current height is:
   _____ pounds

2. What is the least you have ever weighed since reaching your current height? (Do not count any weight losses due to medical conditions or medications). The least I have weighed since reaching my current height is:

   _____ pounds

3. What is your current weight?

   _____ pounds

4. Please determine the difference between your answer to number 1 and number 3. If this difference is less than 5 lbs. skip this item and go on to item 5. If this difference is 5 lbs. or more, indicate which of the three following statements best describe this difference:

   A. The difference between my highest weight and my current weight is due to weight that I lost on purpose.

   B. The difference between my highest weight and my current weight is due to weight I I lost even though I wasn't trying to.

   C. I'm not sure why I weigh less than I once did.

5. For about how long have you been at or close (within 2 lbs.) to your present weight? _____

6. Which of these statements best describe what has happened to your weight during the past 6 months? (circle one)

   A. My weight has stayed about the same
   B. I've been losing weight
   C. I've been gaining weight
   D. My weight has fluctuated a lot

7. Are you currently on a diet? (circle one) Yes No (If no, go to number 9)

8. Are you currently dieting to lose weight or to avoid gaining weight? (circle one)

To lose weight (go to #10) To avoid gaining weight (go to #9)

9. Have you ever been on a diet to lose weight? Yes No
(If no, skip number 10 and 11; you are done)

10. About how long ago were you last on a diet to lose weight? _____

11. About how old were you when you went on your first diet? _____ years old.

12. Please estimate as best you can the number of times in your life you have dieted and purposely lost the amount of weight listed.

How many times in your life have you dieted and lost:

1-4 pounds? ____ times

5-10 pounds? ____ times

11-20 pounds? ____ times

21 or more pounds? ____ times

Hunger Assessment

VERBAL MEASURE OF HUNGER

Please rate the following on a 9-point scale (circle one)

1) How hungry do you feel right now?
1- Not at all 2 3 4 5 6 7 8 9- As hungry as I have ever felt

2) How strong is your desire to eat right now?

1- Very weak 2 3 4 5 6 7 8 9-Very strong

3) How much food do you think you could eat right now?
1- Nothing 2 3 4 5 6 7 8 9- large amount

4) How full does your stomach feel right now?
1-Not at all full 2 3 4 5 6 7 8 9-very full